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LEARNED HELPLESSNESS: DEFICITS ASSOCIATED WITH THE PERCEPTION OF NON-CONTROL AND THE PERCEPTION OF FAILURE

by

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Research carried out at the Institute of Psychiatry,
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MAY 1980

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LEARNED HELPLESSNESS: DEFICITS ASSOCIATED WITH THE PERCEPTION OF NON-CONTROL AND THE PERCEPTION OF FAILURE

The theory of learned helplessness has been formulated to account for the deficits observed after exposure to uncontrollable stimulation. The essence of the theory is that exposure to the dimension of uncontrollability, or response-outcome independence, results in learning which proactively interferes with later learning and results in deficits in emotion, motivation and cognition. There is extensive evidence supportive of the learned helplessness theory from both animal and human experiments, but the theory structure has been reformulated to account for differences in the behavioural repertoires of these organisms, to account for inadequacies of the original theory, and to account for discrepant data. This reformulation has made the theory more elastic and so less amenable to refutation.

It is the aim of this thesis to examine the theory and the evidence critically, and to examine the possibility that the deficits in the human experiments are a function of discontent and dysphoria resulting from the perception of failure inherent in the experimentally defined contingency of uncontrollability. Finally, it will be considered whether the construct of learned helplessness is necessary to explain the behavioural deficits observed in this type of experiment.

Two experimental studies will be reported. The first experiment is a replication of the standard triadic helplessness design but using instructional set and non veridical feedbacks as the independent variables. The second experiment is similar but uses levels of instructional set to facilitate or attenuate the perception of success or failure as independent variables, crossed with the dimension of control and non-control. The experiments involve instrumental pretreatments followed by cognitive test tasks with measures of physiological reactivity, self ratings of affect, motivation etc., and questionnaires relating to trait personality and I.Q. factors.

ACKNOWLEDGEMENTS

The author wishes to thank Irene Martin for her constant advice and encouragement through all stages of this work and the Medical Research Council for their financial support.

The author also wishes to acknowledge the technical support of Les Law, Clive Ioannou, and Ron Bluffield; statistical advice from Robert Baldy; assistance with the testing of subjects from Nona Hemsley and Steve Reszetniak; and the expert typing by Gill Andrews.

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CONTROLLABILITY AND LEARNED HELPLESSNESS

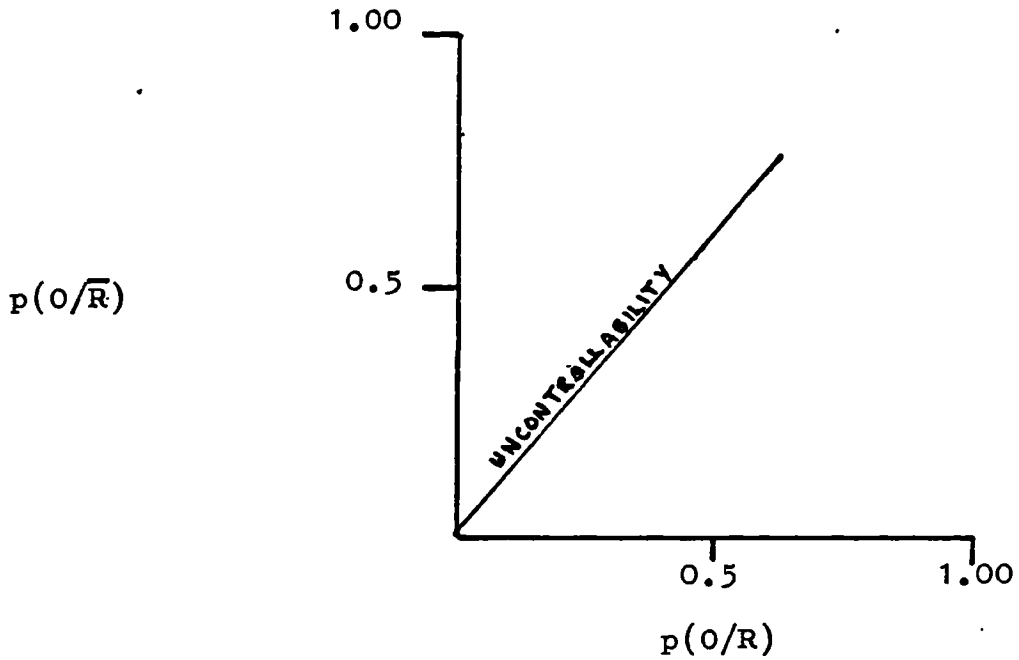
CONTROLLABILITY AND LEARNED HELPLESSNESS

The theory of learned helplessness has been proposed to account for behavioural deficits associated with exposure to uncontrollable stimulation, (Seligman, 1975). Before presenting the theory and citing the evidence in support of the theory, it is necessary to make a distinction between controllability and predictability and also to consider the various definitions of control.

In essence, controllability means that something can be done about an event and predictability means that something is known about that event irrespective of whether something can be done about it or not. In many experiments and in life experiences, predictability and controllability are necessarily confounded. There is a theoretical distinction however, that will be made explicit before considering the various definitions of control.

Given that there is a distinction between classical conditioning and operant conditioning, all possible contingencies that exist between the CS and the UCS, and all possible contingencies that exist between response and reinforcement, can be represented graphically (Figures 1 & 2). For classical conditioning the CS/UCS relationships are represented by the Pavlovian conditioning space (Figure 2). The horizontal axis is the probability of the UCS given the CS and describes the possible schedules of acquisition of the CR. The vertical axis is the probability of no UCS given the CS and describes the extinction schedules of the CR. The diagonal 45^{o} line represents a special CS-UCS relationship termed unpredictability.

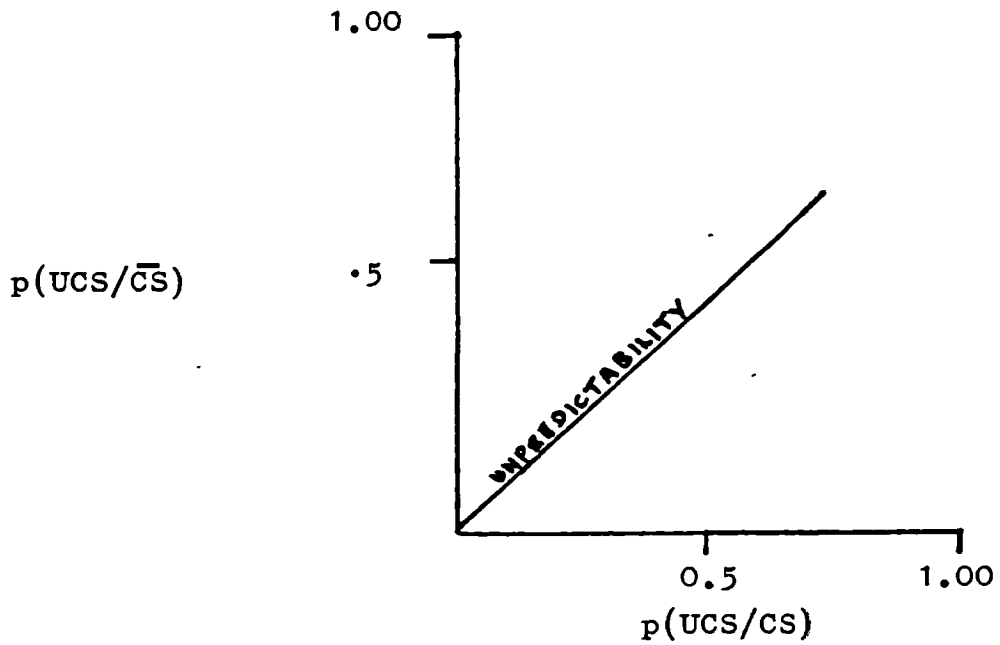
FIGURE 1. The response-reinforcement contingency space



$p(O/\overline{R})$ = conditional probability of an outcome given no response

$p(O/R)$ = conditional probability of an outcome given a response

FIGURE 2. The Pavlovian conditioning space



$p(UCS/\overline{CS})$ = conditional probability of UCS given no CS

$p(UCS/CS)$ = conditional probability of UCS given the CS

From SELIGMAN, 1975.

This is where given the CS, the occurrence or the non-occurrence of the UCS is equiprobable.

$$p\ (UCS/CS) = p\ (UCS/\overline{CS}) = 0.5$$

Uncontrollability is similarly defined by the appropriate contingency in the operant training space that describes response/reinforcement relationships, (Figure 1). The horizontal axis is the probability of the outcome given the response and relates to schedules of reinforcement. The vertical axis is the probability of the outcome given no response and relates to extinction schedules. The diagonal line at 45^{o} is again a special case which is termed uncontrollability. This is where given the response the occurrence or non occurrence of the outcome is equiprobable.

$$p\ (O/R) = p\ (O/\overline{R}) = 0.5$$

The theoretical distinction between predictability and controllability is clear but empirically the effects of these two dimensions are difficult to disentangle. Translating the theoretically defined dimension of uncontrollability into experimental terms we arrive at the following. In an appetitive conditioning experiment (e.g. where food reward is contingent upon bar pressing) the horizontal axis of the operant training space represents all possible schedules of partial reinforcement until the 100% reinforcement schedule is reached where every response results in a food reward. The vertical axis represents all graduations of extinction where no food reward can be obtained irrespective of the number of responses. The 45^{o} axis of uncontrollability is of particular importance to the subject as food reward is equally probable whether the subject presses the bar or not. That is, reinforcement is

independent of responding. Uncontrollability may be defined as response-outcome independence and can be learned by the subject just as any other contingency in the operant training space. The implications of this particular contingency are more evident in an aversive conditioning paradigm.

In an aversive conditioning experiment (e.g. where termination of an electric shock is contingent upon bar pressing) the 45° axis again represents uncontrollability or response-outcome independence. That is, termination of the shock is independent of the subject's responding. The experimental subject has no response available to him with which to control the stimulus and adopts a passivity that is maladaptive and proactively interferes with adaptive learning of response-outcome contingencies when these become available.

There are several types of control that are available to the experimental subject and these will be reviewed.

Definitions of Control

A) Instrumental Control

Here, controllability refers to an instrumental space where the subject is able to make a response that modifies the event. The response can be either active or passive, and can escape (Corah & Boffa, 1970), avoid (Averill & Rosenn, 1972), or attenuate the impact of the stimulus by reducing its intensity or changing its probability (Staub, Tursky & Schwartz, 1971). Perceived instrumental control is a variant of instrumental control and subjects

are induced to believe that they are controlling the event, but they are unable to do so (e.g. Geer, Davison & Gatchel, 1970). Although a seemingly trivial variation it is of importance psychologically as subjects are exposed to identical contingencies but develop different perceptions of these contingencies. It is also important methodologically as it enables both controllable and uncontrollable conditions without the disadvantages of the yoked control design (Church, 1964; Levis, 1976).

B) Self Administration

This is where the subject is allowed to deliver the aversive event to himself (e.g. Ball & Vogler, 1971; Staub, Tursky & Schwarz, 1971). The objective aspects of the situation are not altered as the physical characteristics of the stimulus remain the same whether the experimenter administers it or whether the subject administers it. This is a situation which confounds controllability with predictability, as the subject administered stimulus is always predictable, whereas this is not necessarily the case when the experimenter administers the stimulus. The next case of controllability allows for this.

C) Actual Control equated for Predictability

This is where additional signals are given so that the subject has equivalent information about the predictability of the event whether it is self administered or experimenter administered (e.g. Geer, Davison & Gatchel, 1970).

D) Potential Control

Subjects in this condition are led to believe that some controlling response is available to him but he refrains from using it. Typically, the subject has a "panic button" but he is asked not to press it (e.g. Corah & Boffa, 1970; Glass, Singer & Friedman, 1969).

The above definitions of controllability have been critically evaluated (Miller, 1979) and she proposes that their major common virtue is to hold the physical situations identical between a group with and a group without control, varying only the psychological factors. The major interest in the experimental studies to be reported lies with instrumental control, particularly perceived (but non-veridical) instrumental control. Subjects are induced to believe that they either have control or do not have control but differences in predictability are minimised since the event actually occurs independently of the subject's responding.

Having outlined briefly the concept of control and presented the various definitions of control, the theory of learned helplessness will now be reviewed.

The Theory of Learned Helplessness

It was mentioned previously that exposure to the 45^{o} dimension in the operant training space resulted in deficits in the subject's behaviour. The theory of learned helplessness has been proposed to account for these deficits (Seligman, 1975). Due to problems of satiation the experimental data rely upon aversive stimulation both in the animal and the human literature. This literature will

be reviewed in some detail later. This section will be concerned with presenting the theory and discussing the experimental paradigms employed in the studies.

The basic paradigm employed is the triadic design which uses three groups of subjects. One group receives uncontrollable stimulation by virtue of being yoked to subjects in another group that are able to control the stimuli. This controls for the effect of the stimulation per se, and enables a direct comparison of control vs. non-control. The third group receives no stimulation at all but remains in the experimental room for the same duration as the other two groups. After the training trials all subjects are tested in an experiment where control is available and group differences can be tested. The animal experiments have tended to use electric shock as the aversive stimulus and the human experiments have used shock, aversive noise, insoluble problems etc.

The cornerstone of the theory of learned helplessness lies in the dimension of uncontrollability. The operant training space illustrates that an organism can learn about the probability of an outcome, given a response and similarly that an organism can also learn about the probability of an outcome given that it did not make a response. The new step involved in the learned helplessness theory is that an organism can learn about these probabilities conjointly. That is, when responding and reinforcement are independent, learning takes place. The result of such learning is a special case in that it is maladaptive by having profound consequences for the organism as regards future learning and emotional state. The triadic

experimental design is essential as it enables the discrimination between control and non-control, uncontaminated by the use of aversive stimulation per se.

Seligman (1975) has proposed that the theory has three basic components:-

INFORMATION ABOUT THE CONTINGENCY ⟶ COGNITIVE REPRESENTATION OF THE CONTINGENCY. (LEARNING, EXPECTATION, PERCEPTION, BELIEF) ⟶ BEHAVIOUR

The organism begins with the information about the contingency of outcome upon response which is defined by the experimenter in terms of the experimental paradigm. Secondly, this information must be processed and transformed into a cognitive representation of the contingency i.e. the expectation that responding and an outcome are independent. Thirdly, this expectation is the causal condition for the deficits associated with helplessness. Seligman has emphasised that the second stage of the theory is crucial as mere exposure to information is insufficient - it is possible that an organism can be exposed to the contingency and yet not form such an expectancy and so demonstrate no signs of helplessness deficits. An example of this is immunization which will be described later on.

The essence of the theory is that the dimension of uncontrollability can be learned as any other response/outcome relationship, but this is a special case of learning with respect to the deficits observed after exposure to this contingency. The exact nature of the deficits is as follows:-

A) Motivational Deficits

The expectation that responding will terminate aversive stimulation provides the incentive for the organism to initiate voluntary responses. The learned contingency of response/outcome independence proactively interferes with subsequent learning and response initiation wanes. That is, exposure to uncontrollability produces a motivational deficit characterised by a diminution in response initiation.

B) Cognitive Disturbance

The motivational disturbance leads to a decrease in response initiation. The cognitive disturbance relates to this as learning that an outcome is independent of a response makes it more difficult to learn that responses produce the outcome. After exposure to uncontrollable stimulation it takes longer/requires more trials for the organism to learn that control is now possible. Learning of response outcome independence proactively interferes with later learning of other response outcome relationships.

C) Emotional Disturbance

Given that aversive stimuli cause a heightened emotional state (fear/anxiety), then this fear will occur until the organism learns that the aversive stimuli can be controlled which reduces the fear/anxiety, or, until the organism learns that the aversive stimulation cannot be controlled in which case the fear/anxiety will be maintained until it is eventually replaced by a passivity similar to depression.

In summary, the deficits caused by the learned contingency of response/outcome independence are a reduction in motivation to control the outcome, an interference with

learning that responding controls the outcome, and a fear/anxiety state that develops into a state analogous to depression.

Several alternative theories have been suggested to account for these deficits and these will be outlined. It is worth mentioning two implications of the theory of learned helplessness that have been supported. These are the immunization against and the cure of helplessness.

Cure and Immunization

As the theory of learned helplessness deals with the effects of exposure to uncontrollable contingencies, it would be expected that the theory should make predictions about the prevention and amelioration of such deficits by exposure to controllable contingencies. The theory predicts that the state of helplessness should be alleviated by exposure to contingencies where the outcome is dependent upon responding, but the experimental design does not allow these contingencies to be available to the organism. When helpless animals are forcibly exposed to controllable outcomes (e.g. by forcing the animal to make an escape response in the shuttle box), then it is able to learn the response outcome relationship and the behavioural deficits diminish. Hence, the state of helplessness can be 'cured'.

Also the theory would predict that prior experience of control over aversive stimulation should interfere with the expectation of response outcome independence and inhibit the state of helplessness. There is evidence in support of this prediction (Seligman, 1975), and it seems that organisms can be 'immunized' against becoming helpless by previous

exposure to controllable contingencies.

There are limitations concerning helplessness and these will be discussed at some length when the reformulation of the original theory is reviewed. There are also several alternative theories that have been proposed to account for the behavioural deficits and these will now be considered.

Alternative Theories to Learned Helplessness

The alternative theories that will be considered here are those that deal specifically with the finding that inescapable shock can interfere with later learning of shock avoidance/escape. The experiments using human subjects tend to be more complex and the alternatives are presented at a different level of explanation, so it is more appropriate to deal with these data in the chapter concerning the reformulation of the original theory.

There are three main alternative explanations of the helplessness data which are as follows:-

A. Competing Motor Response Theories

There are three versions of the competing motor response theory but the central issue is that some motor response is learned during the exposure to uncontrollable shock that competes with motor responses necessary for subsequent escape. This avoids the necessity to postulate a cognitive representation of the uncontrollability contingency and has been proposed from a more classical learning theory standpoint.

The mechanisms by which it is suggested that this can occur are as follows:-

1. By superstitious reinforcement. That is, that some motor response occurs at the moment of shock termination and is thus reinforced.
2. That active responses are occasionally punished by the shock onset so that a general passivity is reinforced.
3. That some specific response made by the animal could reduce the severity of the shock.

These explanations are inadequate because there is no evidence of superstitious responding. Specific motor responses are likely to be equally reinforced and punished in the experiment. Also, helplessness has been demonstrated using curarized animals (Overmier & Seligman, 1967) and also where the animal is required to make a passive avoidance response (Maier, 1970).

B. Adaptation and Sensitization

These theories suggest that the animals adapt to the electric shock and so are no longer motivated to respond, or, the animals are so upset by the shock that they are too frantic to organise an adaptive response. This is unlikely as:-

1. There is no evidence of adaptation to intense shock.
2. Adaptation is unlikely to persist over the time course of helplessness.
3. If the theories were adequate then the deficits would occur irrespective of whether the shock was escapable or inescapable.

C. Physiological Approaches

Based on the finding that exposure to inescapable shock causes nor-adrenaline depletion and that escapable shock causes an increase in nor-adrenaline, Weiss et al. (1975), have suggested that nor-adrenaline depletion may be the explanation of the helplessness data. But the crucial experiments have not been replicated (Seligman, 1975), and there are clear disparities between the levels of shock used and other experimental details that makes the comparison of the Weiss data and Seligman's data debatable. It is also an objection that due to the complex relationship between environmental stress and physiological reactivity, a theory that is couched in purely physiological terms and does not recognise the importance of environmental contingencies is unlikely to explain all the characteristics of the helplessness syndrome.

To summarise, this chapter has been concerned with the definition of terms and stating the original theory of learned helplessness. Uncontrollability has been defined as an environmental contingency that can cause the perception of response/outcome independence resulting in deficits that have been termed helplessness.

Alternative theories have been proposed to account for these deficits but in general these theories are unable to account for all the findings. The most convincing theory is physiological and may provide a theoretical substrate for the helplessness phenomenon rather than an adequate alternative.

The evidence from the animal and human literature will be reviewed critically to prepare the ground for the reformulation of the original theory. This reformulation is concerned with the cognitive mediation of the contingencies and is central to the studies to be reported.

CHAPTER 2

EVIDENCE FROM THE ANIMAL EXPERIMENTS

The theory of learned helplessness was originally proposed to account for the learning deficits observed in dogs after prior exposure to inescapable electric shock. An account of the literature on the animal experiments will be given to illustrate the derivation of the theory and also to pinpoint certain differences in the methodology and conceptualisation between the human and the animal studies.

An experimentally naive dog, when placed in a shuttle box, becomes frantic at the onset of the first electric shock and runs around the box until it scrambles over the barrier and escapes the shock. Over trials, the escape behaviour of the animal becomes more organised and the dog eventually waits for the signal for the shock and then jumps the barrier so that it can avoid the shock altogether. If the animal is not experimentally naive, but has received a series of inescapable shocks in a Pavlovian hammock then the animal will behave initially as the experimental animal, that is, it will become frantic and run around the shuttle box. However, it will not acquire the necessary response to escape/avoid the electric shock and the animal becomes passive and seems to give up trying to escape the shock.

This observation, made in 1967 by Overmier and Seligman, led to a series of experimental studies which constitute the evidence for the theory of learned helplessness. There are a number of studies that predate these experiments which may be regarded as examples of helplessness. For example, Richter (1957) found that rats that had been manually restrained showed a shorter latency to death by drowning than rats that had not been exposed to such restraint. Mowrer & Viek (1948), found that rats exposed

to uncontrollable shock showed a greater inhibition of feeding prior to the shock than rats exposed to controllable shock. Similarly, the work on experimental neurosis in animals (Masserman, 1943) may be regarded as being due to deficits associated with non-control contingent upon impossible discrimination tasks. Even though the Mowrer & Viek results were interpreted as being due to "fear from a sense of helplessness", the controlled studies and the development of the theory were carried out by Seligman and his co-workers.

As the original experiments were carried out on dogs, this work will be described first. The standard procedure that produces learned helplessness in the dog is as follows:-
(Overmier, 1968; Overmier & Seligman, 1967; Seligman & Groves, 1970; Seligman & Maier, 1967; Seligman, Maier & Geer, 1968).
On the first day the dog is strapped into a Pavlovian hammock and given 64 inescapable electric shocks each of 5.0 seconds long and of 6.0 mA intensity. The shocks are unpredictable and are temporally random. Twenty-four hours later the dog is given 10 trials of signalled escape/ avoidance training in a two way shuttle box where the necessary response is to jump the barrier separating the two halves of the box. Shock can occur in either side so that there is no one place that is safe, but the response of shuttling alone can terminate the electric shock. The signal for trial onset is light dimming and the signal stays on until the trial is over. There is a 10 second interval between the onset of the signal stimulus and the

onset of the electric shock. If the dog jumps the shoulder high barrier the shock is escaped/avoided and the signal terminates. Failure to escape/avoid results in a 4.5 mA shock of 60 seconds duration.

Over the years between 1965-1969, 150 dogs were trained and tested and it was found that about two-thirds of these failed to learn. One-third of the dogs behaved in the shuttle box as the experimentally naive animals and this has been explained in terms of immunization which will be described later. The dogs that had received the inescapable pretreatment appeared to be physically capable of jumping the barrier. Also, they occasionally jumped the barrier between test trials and readily escaped through the entrance door if this was left open during the test session. These observations lead to the conclusion that the learning deficits are of a psychological nature. Variations on the basic paradigm have included using various levels of shock intensity, shocks of different duration and different frequency, signalled and unsignalled shock, and also using the Pavlovian hammock and the shuttle box interchangeably.

Similar results have been reported for cats (Masserman, 1943, 1971; Seward & Humphrey, 1967; Zielinski & Soltysik, 1964). The study by Thomas & Balter (1974), reports an effect which is apparently identical to the learned helplessness effect in dogs. Cats given inescapable shock in a hammock subsequently failed to escape shock and passively accepted the shock as did the dogs in the studies by Seligman et al. Fish also show learning deficits after exposure to inescapable shock. Padilla, Padilla, Ketterer & Giacolone (1970), gave inescapable shock to goldfish and

then tested them in an aquatic shuttle box. These fish were slower to respond than the naive controls. Other related studies have been reported (Behrend & Bitterman, 1963; Bintz, 1971; Frumkin & Brookshire, 1969; Padilla, 1973).

The learned helplessness effect has been difficult to establish in the rat. Rats given prior inescapable shock were typically slower to respond on the first few trials, were slower to acquire the avoidance response, but did not fail to learn (Maier et al., 1969; Seligman et al., 1971). It seems that learned helplessness can be shown in the rat given that the response necessary to terminate shock in the test task must be difficult, and a response that the rat does not readily perform, (Maier, Albin & Testa, 1973; Maier & Testa, 1975; Seligman & Beagley, 1975; Seligman, Rosselini & Kozak, 1975). So, if rats are exposed to inescapable shock and then tested on a simple escape response such as a single bar press (FR1) or a single shuttle in a shuttle box, no deficits are found. FR1 means that there is a Fixed Ratio between the number of responses required to cause reinforcement. In this case reinforcement is contingent upon one response made by the subject. If the response requirement is increased (i.e. an FR3 schedule for bar pressing, that is, reinforcement occurs when the subject has made three bar presses or an FR2 schedule for shuttling, that is, from one end of the shuttle box to the other and back again) then the rat that has received the inescapable pretreatment fails to escape.

All of the deficits observed in the animals pretreated with inescapable shock are in comparison with animals

previously exposed to escapable or no shock conditions, and these animals readily learn even more difficult response schedules.

These results have also been extended to developmental studies. Hannum, Rosellini & Seligman, (1976), found that inescapable shock given to rat pups just after weaning produced deficits 90 days later on an FR3 bar press escape task, compared to rats that had been given no shock or escapable shock just after weaning.

It has also been reported that mice that have been exposed to inescapable electric shock demonstrate learning deficits, (Braud, Wepman & Russo, 1969).

The evidence cited so far provides considerable support for the existence of motivational deficits that result from prior exposure to uncontrollable stimulation. There is evidence to suggest that this deficit is not task specific but reflects a more generalised organismic debilitation. For example, Braud et al (1969), report an experimemt in which mice were required to climb a pole to escape electric shock. This group of animals was yoked to a group where no response was available to escape the shock, and a third group received no shock at all. It was found that the yoked group was poorest at escaping from an underwater alley. Rosellini & Seligman (1975) found that rats previously exposed to inescapable shock failed to escape from the goal box in a frustrative non-reward task compared to rats exposed to escapable shock or no shock conditions, which readily escaped. It has also been found that rats previously exposed to inescapable shock are less aggressive than rats that had received escapable or no shock in a shock-elicited

aggression situation (Maier et al, 1972). Seligman & Maier, 1967, report unpublished data that suggest that dogs were less competitive in feeding if they had been exposed to inescapable shock as pups.

It seems that helplessness can occur across a variety of pretreatments and test tasks. However, all the studies cited so far have involved aversive stimulation, whilst the theory predicts that helplessness should occur as a function of any uncontrollable pretreatment. The main problem with experiments using appetitive reinforcement is that of satiation, i.e. the animals do not remain sufficiently motivated for a long enough time period to allow sufficient exposure to the contingencies necessary to induce helplessness. However, Engberg, Hansen, Welker & Thomas (1973) found that pigeons previously exposed to food delivered independently of responding showed slower acquisition when tested on a key peck autoshaping procedure compared to pigeons whose food delivery was response contingent, or pigeons which had received minimal magazine training. This particular experiment has been criticised by Gamzu, Williams & Schwartz (1973) as :-

1. autoshaping is largely controlled by Pavlovian contingencies (Moore, 1973) and helplessness is the debilitation of instrumental responding.

2. the results can be explained by a competing motor response theory, due to the similarity of treadle responding and standing on the grain hopper.

However, Engberg et al (1973), removed the treadle during the autoshaping task and a further investigation

(Welker, 1974) which included the key during the treadle test rules out the competing motor response theory.

Bainbridge (1973), gave 50 day old rats solvable discrimination problems, unsolvable discrimination problems, or no problems at all. The rats were tested 20 days later with the same tasks (but using different stimuli), or in a Hebb-Williams maze based on spatial cues. The rats that had been given the unsolvable problems performed very poorly in both the same and the different apparatus.

The evidence so far suggests that exposure to uncontrollability produces a motivational deficit. Maier & Seligman (1976) have proposed that such exposure produces a psychological state which undermines response initiation quite generally, i.e. across species and across tasks. There is clearly a limit to the generality of the helplessness phenomenon and also a variety of other possible explanations, but there are substantial supportive data. Exposure to uncontrollability causes a deficit in the perception of subsequent control which Seligman has called a cognitive deficit. That is, experience with uncontrollable events produces a difficulty in learning that responses are now effective in controlling the stimulation. Usually, once a naive dog has made a successful escape from the electric shock, it will readily learn to escape/avoid future shocks. Dogs previously exposed to uncontrollable shock do sometimes make a successful escape and then, instead of learning rapidly, they revert back to passively accepting the shock. Whether a 'cognitive' explanation is appropriate to the animal experiments is debatable. An explanation in terms of habit strength or even that such effects are

epiphenomena of the motivational deficit may be sufficient. The evidence from the human experiments is more supportive to the existence of a distinct cognitive deficit and this will be reviewed later. However, it does seem that CS-UCS independence is an actively learned contingency (Mellgren & Ost, 1971; Kemler & Shepp, 1971; Thomas et al, 1970; MacKintosh, 1973; Alloy & Bersh, 1979). For example, if nothing was learned after exposure to the CS-UCS independent contingency then no behavioural consequences would be expected. MacKintosh (1973), has shown that subjects can be trained to ignore stimuli that do not predict reinforcement. Similarly, the learned helplessness experiments clearly show behavioural consequences after exposure to uncontrollability.

There is also considerable evidence that the dimension of uncontrollability has profound emotional consequences. Overmier & Seligman (1967) found that dogs pretreated with uncontrollable electric shock showed helplessness deficits at 24 hours but these deficits remitted after 48, 72, and 128 hours. This time course suggests that there is an emotional component to the debilities. The executive monkey study (Brady, Porter, Conrad & Mason, 1958), showed that monkeys that were able to control electric shock developed ulcers and died. Monkeys that were yoked to these animals and received the identical electric shocks but were unable to control the shocks did not show any unduly adverse effects. This result is contrary to the predictions from learned helplessness theory, which predicts that the yoked monkeys that had received the uncontrollable electric shocks would show the debilitation. Weiss (1972) has suggested that this finding may be an artifact of the

way in which animals were allocated to experimental groups. The more emotional a monkey is, the more quickly it begins to bar press when shocked (Sines, Cleeland & Adkins, 1963). As the executive animals were chosen on the basis of being first to acquire the bar press response, their debilities may be a function of greater emotionality per se and a greater susceptibility to shock. The emotional consequences of the dimension of uncontrollability have been investigated more extensively in rats.

Weiss (1968, 1971a, 1971b, 1971c), replicated the executive monkey study using rats. Animals were randomly assigned to three groups in a triadic design involving controllable shock, uncontrollable shock, and no shock conditions. The executive animals showed fewer and less severe ulcers than the yoked animals. Also the yoked animals lost more weight, defaecated more, and drank less than the executive animals. Exposure to uncontrollable shock causes more stress/emotional debilitation in terms of gastric ulceration than exposure to controllable shock.

Hearst (1965), found that exposure to uncontrollable shock resulted in the breakdown of a well trained appetitive discrimination in rats, whilst the discrimination was maintained where the shock was controllable. Several other studies, although they do not fit directly into the learned helplessness paradigm, reflect the debilitating results of uncontrollability. For example, the Shenger-Krestnikova study showed that dogs became distressed when overfine discriminations were required to gain food reward (Pavlov, 1927). The work by Liddell, James & Anderson, (1934), which showed that sheep exposed to uncontrollable

electric shock developed maladaptive behaviours also illustrates the emotional consequences of uncontrollability. These data are supportive to the theory of learned helplessness, if only in a post hoc rationalisation.

The alternative theories that have been proposed to account for these data have already been outlined in brief and a more complete examination will now be given.

Alternative Explanations of the Animal Helplessness Data

The alternatives can be classified into two main categories of motivational theories and motor theories, Maier & Seligman (1976).

Motivational Theories

a) Adaptation

This explanation proposed that a subject adapts to shock during pretreatment and so is not sufficiently motivated to escape shock on testing. This explanation is clearly inadequate on several counts:-

1. adaptation to repeated intense shock has never been demonstrated (Church, Lolordo, Overmier, Solomon & Turner, 1966).
2. it is unlikely that such adaptation could persist for the time course of 24 hours +.
3. the animals do not behave as though they are adapted to the shock.
4. raising the shock level on testing does not eliminate the interference (Overmier & Seligman, 1967).
5. adaptation does not account for the lack of deficits in the escapable shock conditions.

6. adaptation cannot handle the phenomenon of immunization by exposure to controllable shock prior to the uncontrollable pretreatment.

7. adaptation cannot explain the cure of the deficits by forcible exposure to controllable contingencies.

b) Sensitization

Essentially the opposite of the adaptation hypothesis, i.e. that shock sensitizes the subject so that it is too highly motivated to organise responses. This explanation is also clearly inadequate.

1. sensitization does not explain the absence of responding and the passive acceptance of the shock.

2. Overmier & Seligman (unpublished data), found that lowering the shock level on testing did not attenuate the interference.

3. points 5, 6 and 7 of the argument against adaptation also apply to the sensitization explanation.

Motor Activity Theories

These hypotheses propose that exposure to uncontrollable electric shock interferes with subsequemt escape because it changes motor activity.

a) Incompatible Motor Response Theories

Proposed by Bracewell & Black (1974) who suggested that it is the explicit punishment of movement which produced a retardation in shuttle box acquisition. This does not explain why escapable shock does not produce the same deficits as inescapable shock. The evidence cited by Bracewell & Black is questionable as it is not clear whether the dimension of controllability was manipulated.

Another variation of the incompatible motor response theory has been suggested by Anisman & Waller (1973). They argued that exposure to shock induces response repertoire changes in the organism, and if no coping response is available, 'freezing' becomes the organism's dominant reaction to shock. The evidence cited by Anisman & Waller consists of studies of avoidance and not escape. The subjects in the experiments carried out by Seligman do not seem to freeze, and the theory fails to explain the differences between escapable and inescapable shock. Maier & Seligman (1976), cite further evidence against both these variants of the incompatible motor response theory:-

1. rats become helpless when the escape response is an FR2 response, but do not become helpless so readily if the response is an FR1 (Maier et al, 1973). So it seems that an FR1 response is an initial reaction to shock and is clearly not a freezing response.

2. the theory cannot explain the evidence from the immunization studies. If the theory was sufficient then the deficits would occur even after the controllable immunization phase.

3. the training of a response directly incompatible with shock escape did not duplicate the effects of inescapable shock (Maier, 1970).

4. the Bracewell & Black version of the theory predicts that passivity may reduce the pain of the shock or that other motor responses reduce the pain of the shock. However, Overmier & Seligman (1967), found that paralysis by curare during the pretreatment phase of the experiment did not prevent or reduce the

deficits, so the prediction that if movement is prevented the deficits would not occur is not supported.

5. Rosellini & Seligman (1975) found that inescapable shock interfered with goalbox escape but did not interfere with runway acquisition and extinction, which would be expected with the incompatible motor response theory.

6. these theories are specific to experiments using electric shock. It is unlikely that such explanations can account for deficits due to unsolvable discrimination problems or loud noise as an aversive stimulus.

b) Motor Activation Deficit Hypothesis

This explanation has been proposed by Weiss et al, 1975. The core of the theory is that deficits are due to nor adrenaline depletion caused by severe inescapable electric shock, and so the subjects were unable to co-ordinate sufficient motor activity to perform adequately. Miller & Weiss (1969), argued that the time course of helplessness in the dog (48 hours), suggests a time based physiological change rather than learning, which should persist longer. Also central catecholamines have a time course following depletion (Rech, Bowys & Moore, 1966) and are involved in the mediation of movement (Hermann, 1970). Weiss, (1968, 1971a, 1971b, 1971c), has shown that rats given inescapable electric shock undergo greater stress than rats given equivalent escapable shocks as measured by gastric lesions, loss of body weight, plasma steroid levels, and fearfulness. Weiss, Stone & Harrell (1970), found that rats exposed to inescapable shock had lower nor adrenaline levels in the

brain than rats exposed to escapable electric shock. So, Weiss (1975), proposed that inescapable shock produces central nor adrenaline depletion that depresses the movement of the organism. Weiss (1975), cites 12 experiments supportive to this interpretation of the data, but Maier & Seligman (1976), have criticed the logic and data underlying this theory. Firstly, the assumption that the time course favours a physiological explanation is highly debatable as there is evidence of learning retention losses over short periods of time (D'Amato, 1973; Spear, 1973). The time course has been shown to be variable and can extend to 7 days in the dog (Seligman & Groves, 1970), and in the rat (Seligman & Beagley, 1975). The discrepancies between the experimental paradigms employed also preclude the comparison of Seligman's data and Weiss' data. In all but one of the nor epinephrine studies, Weiss used a minimum of 20 hours inescapable shock at a minimum level of 3.0 mA, which is far more severe than that used by Seligman (1 hour at 1mA). Nor adrenaline assay was performed immediately after the inescapable shock session with the exception of one experiment (Weiss et al, 1970c exp. 1) in which the group of rats given inescapable electric shock did not show nor adrenaline depletion. Also, Weiss et al (1975) report that the nor adrenaline depletion was only evident if the animals were group housed prior to the experiment, all the animals in Seligman's studies were individually housed. Seligman cites grounds for questioning each individual experiment and makes several other points which cannot be accounted for in terms of the motor activation deficit hypothesis.

1. Maier & Testa (1975), found that although inescapably shocked rats were poor at learning FR2 shuttling they will learn as well as controls if there is a brief break in the shock following the first response. The motor activation deficit hypothesis cannot explain why the behavioural deficits are so sensitive to stimulus contingencies.

2. Immunization is accounted for in terms of the animal having reduced the amount of motor behaviour required during testing as it does not have to search for the correct response. However, immunization occurs even when different responses are required.

3. The hypothesis cannot account for the alleviation of the deficits by forcible exposure to controllable contingencies.

4. The time course of helplessness is variable and can extend beyond the time expected if the deficits are due to nor adrenaline depletion.

5. Hannum et al, 1976, found that rats given four sessions of inescapable shock after weaning failed to learn as adults. This did not occur if the shocks were escapable and an explanation in terms of permanent nor adrenaline depletion is unlikely.

6. The data cited by Weiss et al (1975), deal only with the rat and dog experiments using electric shock. It cannot account for the experimental data using unsolvable problems or human subjects.

It is clear that the only explanation that provides a plausible alternative to the theory of learned helplessness

is the motor activation deficit hypothesis and as Maier & Seligman (1976), have made explicit, this theory is limited to the rat experiments and there are striking differences between Weiss and Seligman in the experimental paradigms employed. Considering the cross species and inter task generality of the theory of learned helplessness, this theory remains the most adequate explanation of the experimental data.

Maier & Seligman (1976), note several points that the theory of learned helplessness cannot account for, and these are:-

1. The time course has yet to be satisfactorily explained. One shock session produces a temporary effect in dogs and four sessions produce a more permanent effect. There is evidence to show that learned helplessness may be permanent in rats after one session of inescapable shock. These effects may be due to experience with controllable contingencies prior to the inescapable pretreatment; however there are insufficient data to establish this as yet.

2. The finding that helplessness deficits can be demonstrated in rats only when an FR2, or even a more complex response is required, is a further complication. This may be a function of the amount of physical effort involved, but Maier & Testa, (1975), found no deficits on a FR2 schedule if there is a break in the shock after the first response. They also found that deficits do occur on an FR1 schedule if shock termination is delayed. It is possible that the test tasks employed so far involve a large amount of

intrinsic feedback that attenuates the influence of the perception of response outcome independence on the behaviour of the animal.

3. The theory does not specifically predict whether deficits will be shown on an aversive test task given an uncontrollable appetitive pretreatment. Nor does it predict transfer across Pavlovian/operant phases of the experiment.

4. The conditions under which the perception of response outcome independence can occur in human subjects has yet to be specified. This is important as given the greater behavioural repertoire of the subject, objective response outcome independence may not result in the perception of response outcome independence and similarly, the perception of response outcome independence can also occur when the outcome is contingent upon the subjects response. This will be dealt with in depth when the reformulation is discussed in the next chapter.

More recent research reveals that learned helplessness in animals is a complex phenomenon and that there are many issues yet to be resolved. With respect to the third point mentioned above, namely the generalisation across qualitatively different pretreatment and test phases of the experiment, Garber et al. (1979) have found that learned helplessness persists from the initial pretreatment of weanling rats to test phases in the adult. Further, generalisation does occur widely and has been shown to occur across aversive/appetitive pretreatment and test sessions.

This is supportive to the finding by Rosellini (1978) that inescapable shock interferes with the acquisition of an appetitive operant. However, Rosellini and Seligman (1978) demonstrated generalisation with difficulty, that is, generalisation only occurred if the shock intensity used on the test task was the same as that used in the pretreatment task.

There are even difficulties in the replication of the basic learned helplessness paradigm. For example, Beatty (1979) found no evidence of learned helplessness in rats using inescapable foot shock and an FR 3 instrumental test task. Whilst learned helplessness can be demonstrated and has been demonstrated repeatedly in a variety of organisms it seems clear that there are many underlying features to learned helplessness which have yet to be explored in detail.

Jackson et al. (1979) have found that rats exposed to uncontrollable shock show a temporary analgesia to shock. After 24 hours this analgesia dissipates but can be restored by application of a brief shock. Whilst there is considerable evidence against an habituation explanation of learned helplessness, this finding poses the possibility that learned helplessness deficits may be, at least in part, a function of temporary analgesia to shock. This seems unlikely given the observable behaviour of rats in the test session but given the small differences observed between rats that have received controllable shock to those that have received uncontrollable shock, this analgesia may be sufficient to account for learned helplessness deficits observed in some studies.

A study by Childress and Thomas (1979) poses another interesting problem for learned helplessness. They found that dorsal mid-brain stimulation does not cause learned helplessness deficits. This implies that peripheral aversive stimulation may be a necessary feature for helplessness to occur which is a problem for a strict cognitive model of learned helplessness. That is, if deficits are due to the perception of response outcome independence these deficits should occur whenever uncontrollable outcomes are perceived irrespective of whether the stimulation is peripheral, e.g. foot shock or central, e.g. direct stimulation of the brain.

As well as the site of stimulation the nature of the stimulation both qualitatively and quantitatively is also an important feature. Lawry et al. (1978) varied certain characteristics of the shock. For example, AC versus DC and continuous versus pulsatory shock in all possible combinations across pretreatment and test phases of the experiment. They found that only certain combinations caused learned helplessness deficits. Alloy and Bersh (1979) found that previous experience with control over shock intensity, not shock per se, was sufficient to prevent learned helplessness deficits when the animals were put through the standard learned helplessness paradigm. It seems to be an anomaly that whilst some workers in the field have demonstrated wide generalization across tasks, others have found minutiae of the actual treatments to be significant determinants of the deficits.

One issue that has been examined in greater detail relates to the involvement of motor activity during the pretreatment phase and the possibility that part of the learned helplessness deficit may be due to learned inactivity. Intuitively this is a plausible component as any response may be conceptualised as being punished by the advent of the electric shock by the very nature of the fact that it is uncontrollable. Frank (1977) used a stabilometer to check activity differences between rats exposed to either controllable or uncontrollable shock found no apparent differences between conditions. Jackson et al. (1980) point out that previous studies aimed at the issue of motor activity have confounded an associative deficit with decreased activity. Four experiments are reported using rats in a Y maze task in which reduced activity caused a slower response whilst an associative deficit caused an incorrect choice. Hence, decreased activity may be differentiated from an associative deficit. The results provide clear support for the prediction from the theory of learned helplessness that inescapable shock produces an associative deficit.

In contrast to this study, Irwin et al. (1980), report a series of experiments which combined inescapable shock with various temperatures of water which directly affect the level of motoric activity. Water escape testing, when conducted in warm water, was not disrupted by an inescapable preshock. Disruption occurred in colder water, but colder water failed to discriminate between those animals that had received escapable or inescapable preshock. The authors conclude "that inescapable shock results in deficits of

response maintenance but probably has a minor, if any, influence on cognitive/associative processes".

Even though the methods employed in these studies are appropriate and apparently sound methodologically, the issue remains to be resolved. The problem seems to be the way in which inactivity is operationalised in each experiment and it may be that a standard helplessness experiment involves both learned inactivity and a learned associative deficit. In which case, manipulations of the experimental parameters may accentuate one component at the expense of the other. This would lead to different results depending on the manipulation. Putting these studies into perspective with other studies, (including human studies), the evidence does suggest a distinct associative deficit. The extent to which learned inactivity is a significant component of animal studies needs to be evaluated further, if a crucial experiment can be designed. It may be that these factors are inextricably related.

CONCLUSION

It is clear that inescapable pretreatments may be differentiated from escapable pretreatments in terms of the behavioural consequences of such pretreatments. In general, deficits in learning are caused by inescapable pretreatments.

The theory of learned helplessness proposes that these deficits occur as a result of the organism having learned that responses are independent of outcomes, that is, an associative deficit. Various other explanations have been proposed which are largely unsupported in the light of all the data. However, whilst there is good evidence for such an associative deficit there is a possibility that temporary analgesia to shock may be an influential feature and further, that the role of motoric activity needs to be evaluated further. That is, is a motoric deficit a central causal component of helplessness, or, are there particular circumstances which lead to an associative deficit in one case, and to a reduction in motoric activity in another case ? Recent research suggests that whilst deficits due to uncontrollable stimulation can be reliably obtained, the relative contributions of these factors is often confounded.

The theory of learned helplessness which centres around the concept of the perception of response outcome independence remains the most adequate interpretation of the data so far. But there are complex theoretical issues which need to be resolved, e.g. the role of learned inactivity. An associative deficit needs to be established definitively and experiments need to be designed to investigate anomalous

results and examine in greater depth the conditions under which generalisation does and does not occur. It is possible however that many aspects of helplessness are inseparable experimentally and that the crucial experiments are not available.

CHAPTER 3

EVIDENCE FROM THE HUMAN EXPERIMENTS

The theory of learned helplessness has been outlined in the first chapter and the animal literature reviewed in Chapter 2. This chapter is concerned with the literature on experiments with human subjects, a consideration of the differences between the animal and human experiments, and a review of the reformulation of the original helplessness theory.

The animal experiments have typically involved the use of the triadic experimental design, in which subjects are randomly assigned to one of three experimental groups:-

a) a group that receives a controllable pretreatment, for example, escapable electric shock.

b) a group that receives an uncontrollable pretreatment, for example, inescapable electric shock.

c) a group that receives no pretreatment at all.

All subjects are then tested on a different kind of task and the characteristic deficits are observed in the group that had received the uncontrollable pretreatment.

The human experiments are similar in so far as the triadic experimental design has often been used, but the nature of the pretreatment tasks and the test tasks have been more varied. The tasks have involved escaping/avoiding loud noise, electric shock; attempting to solve discrimination problems, anagrams etc. An example of a fairly typical learned helplessness experiment using human subjects is a study by Hiroto (1974). Using the triadic experimental design subjects in the escape group received loud noise which they learned to turn off by button pushing. The subjects in the inescapable group received the same noise

but their responses were ineffective in terminating the noise. A third group of subjects received no noise at all. All subjects were then tested on a hand shuttle box. That is, subjects had to move their hands from side to side in order to escape the noise. Both the no noise and the escape groups learned readily to shuttle with their hands, but the inescapable group failed to escape or avoid. Subjects in this group tended to sit passively and accept the aversive noise. These results parallel the results from the original research on dogs, but Hiroto incorporated two other factors in his experimental design that illustrate that the situation is more complex for human subjects. Half the subjects in each of the three groups were told that how they did on the shuttle box was a test of skill, the other half were told that how they did was a matter of chance. Those subjects who received the chance set instructions tended to respond poorly in all groups. Hiroto also varied the personality dimension of "external and internal locus of control of reinforcement" (Lefcourt, 1966; Rotter, 1966), so that half the subjects in each group were internals and half externals. An external is a person who believes, as shown by his responses on a personality inventory, that reinforcements occur in his life by chance or luck and are beyond his control. An internal believes that he controls his own reinforcers and that these are a function of his own skills. Hiroto found that externals became more helpless in his experiment than internals. Hiroto concluded that the experience of uncontrollability with appropriate instructional/cognitive set and with an external personality contributed to the perception of response outcome independence which is the

crucial precursor of the state of helplessness.

This experiment is clear evidence that helplessness can be shown in humans, but it is evident that the context within which the pretreatment phase occurs and also underlying personality dimensions must be considered. The behavioural repertoire of the human subject exceeds that of other species and this must be allowed for by extending the theory to account for these differences, and also accepting that anomalies to the theory will almost certainly arise.

Similar experiments have been carried out and are largely supportive to the finding that learning deficits occur after exposure to uncontrollable pretreatments (Glass & Singer, 1972). The helplessness effect has been shown to generalise across tasks in humans. Hiroto & Seligman (1975) and Miller & Seligman (1975) explored the transfer of helplessness between instrumental tasks and cognitive tasks. Subjects either received escapable, inescapable, or no noise pretreatments and then they were tested on a non aversive anagram solving task. It was found that subjects who had received inescapable noise performed worse on the anagram solving task compared to the escape and no noise groups.

Helplessness has also been demonstrated where the pretreatment phase does not involve aversive stimulation. Hiroto & Seligman (1975) and Klein et al (1976), gave three groups of subjects four sets of solvable, unsolvable, or no discrimination problems. All groups were then tested on the hand shuttle box with loud noise. The group exposed to unsolvable problems failed to escape the noise, whilst subjects who had solvable discrimination problems or no

previous problems readily escaped the noise.

So far, the experiments have shown that the characteristic motivational deficit can be demonstrated after exposure to uncontrollability. Several experiments illustrate that helplessness has a cognitive component i.e. experience with uncontrollability may produce a difficulty in learning that responses have succeeded, even when responding is actually successful. Miller & Seligmam (1975) and Klein et al (1976), report an experiment in which three groups of subjects received escapable, inescapable, or no loud noise. All subjects then attempted two tasks, a task of skill and a task of chance. The skill task consisted of ten trials in which subjects were required to sort 15 cards into 10 categories of shape within 15 seconds. The experimenter arranged to have them succeed or fail on any given trial, so that they were exposed to a prearranged run of successes and failures. At the end of every trial subjects rated what they thought their chances of succeeding on the next trial would be. Subjects exposed to inescapable loud noise showed very little change in their expectancy following success or failure. That is, they had difficulty perceiving that their response would affect their performance. No differences in expectancy were found on a similar chance task, and the other groups rated changes of expectancy congruent with their performance. Similarly, it has been found that subjects previously exposed to inescapable noise require more trials to reach a criterion on the anagram solving task, more evidence that exposure to uncontrollability produces the cognitive set that inhibits the perception of response contingent outcomes.

The evidence for emotional deficits associated with uncontrollability is not so clear cut. Firstly, the exact dependent variables that reflect emotional activity pose a problem. This has been reviewed extensively by Lader (1975), Averill (1973), and Miller (1979). The major physiological variables associated with increases in anxiety are an increase in skin conductance level, more phasic electrodermal activity, and attenuation of habituation of the skin conductance response. Heart rate tends to increase and there is also a tendency to breathe more rapidly and more shallowly. For depression the reverse holds, that is, a lower skin conductance level, less phasic electrodermal activity, deep slow respiration, but there is little evidence for changes in heart rate. A more complete examination of this issue is given in Chapter 5.

As learned helplessness has been proposed as a theory of reactive depression, Seligman (1975), has suggested that exposure to uncontrollability causes an increase in anxiety/arousal which eventually results in depression. This raises the second problem. As long as there is a change in the physiological dependent variables across the experiment this can be accounted for by the theory. That is, an increase in skin conductance level could be due to greater anxiety, and decreased skin conductance level due to the onset of depression. The third problem is an extension of this. That is the actual studies that have used physiological dependent variables.

Bowers (1968), reports an experiment comparing groups of subjects who either controlled or did not control electric shock and concludes that the skin conductance data were 'unhelpful'. Glass & Singer (1972), found no significant differences for skin conductance level between groups that either controlled or did not control electric shock. Geer et al (1970), in a similar experiment found that subjects who did not have control over the electric shock habituated more slowly as measured by skin conductance level. Houston (1972), reported no significant heart rate differences between groups exposed to controllable or uncontrollable electric shock. Glass & Singer (1972), found no differences between control and non-control groups on skin conductance level, nor any evidence of habituation. Gatchel & Proctor (1976), in a more detailed examination of the physiological correlates of helplessness reported the following from a triadic design using noise as an aversive stimulus:-

1. the escape group showed a gradual increase in skin conductance level compared to the non-escape and control group.
2. there was less habituation of the skin conductance response amplitude for the escape group.
3. there was less spontaneous electrodermal activity for the escape group.
4. the escape group maintained a higher mean heart rate level than any other group.

Gatchel et al (1977), replicated the previous study including depressed groups and found that the escape group showed a higher skin conductance level compared to the other groups. Also, they found less habituation of the skin conductance amplitude for this group. Pennebaker et al (1977), found that for a task using control or non-control of aversive noise there were no significant differences between groups on the amplitude of the skin conductance response or for spontaneous electrodermal activity. All groups habituated.

It is unclear how to interpret these results. They are not consistent and the differences in recording techniques and analysis preclude the direct comparison of these studies in any case. The physiological predictions for the experimental studies to be reported are made explicit in Chapter 5. It would be expected that the non-control subjects should become more anxious (higher skin conductance levels and more spontaneous electrodermal activity, perhaps higher mean heart rate levels) and that they might become depressed as the experiment progresses (i.e. lower skin conductance levels, less spontaneous electrodermal activity, lower heart rate). These predictions are based on a fairly inconsistent literature (Averill, 1973), and do not seem to be supported consistently in the helplessness literature to date.

The other main measure of emotionality, self report, has provided fairly consistent evidence that exposure to uncontrollable aversive stimulation is evaluated more negatively than exposure to controllable aversive stimulation, (Miller, 1979).

The data so far suggest that helplessness can be produced in human subjects, and that motivational, cognitive, and emotional concomitants are evident. However, there are a number of studies that are not supportive to the theory of learned helplessness and also several criticisms have been levelled at both the theory and the data.

A study carried out by Thornton & Jacobs (1971), obtained results that did not confirm the learned helplessness theory. The experiment involved a pretreatment of electric shock and a subsequent test of intellectual performance. Subjects who had received the inescapable electric shock during the pretreatment phase significantly increased their scores on the mental ability test. The scores of the subjects who had received escapable shock or no shock remained unchanged.

The authors explained this as being due to the distinct difference between the pretreatment and the test task. That is, the difference was too great to allow the state of helplessness to generalise.

Shaban & Welling (reported in Glass & Singer, 1972, pp. 122-130) have also obtained results which would not have been predicted from the theory of learned helplessness. Two groups of subjects were employed, one group who perceived that their bureaucratic problems were due to forces beyond their control (i.e. the external impersonal system), while the other perceived that their problems were due to forces potentially within their control (i.e. the experimenters). Both groups were denied control by being unable to escape the bureaucratic problems. The behaviour

of the first group was docile, passive, and compliant in a subsequent test session, whilst the behaviour of the second group was reactive, hostile, and negativistic. This was interpreted as being due to a state of helplessness in the first group, while the second group was attempting to re-establish and exercise control over events. The group differences were thought to be due to differences in expectancies for control.

Roth & Bootzin (1974), found that subjects who were administered random reinforcement for their performance on concept learning tasks initiated more controlling behaviour over an aversive event in a subsequent problem solving test than subjects who had received contingent reinforcement or subjects who had received no pretraining at all.

Roth & Kubal (1975), have suggested that there is an underlying curvilinear relationship between experience of non-control and helplessness type behaviour. They support this by noting that Krantz et al (1974), found increased manifestations of helplessness with increased helplessness training. An experimental study involving three levels of helplessness training crossed with two levels of task importance, showed that facilitation and helplessness effects were found, and that task importance and amount of training increased the likelihood of helplessness effects.

This implies that limited exposure to uncontrollability would cause the subjects to become more anxious and it might be expected that they would become more highly motivated to do well on the test task. After prolonged exposure to the dimension of uncontrollability, subjects would become depressed and then show the characteristic

helplessness deficits. So, there is an inverse U relationship between the importance and amount of training with the expected behavioural consequences of such exposure.

Although this explanation is a distinct possibility, this underlying curvilinear relationship is unfortunate as it renders the theory irrefutable. Both facilitation and helplessness effects can now be accommodated. However, there are results which are not in line with the predictions from the original formulation of the learned helplessness theory. The Shaban & Welling study together with the Roth & Kubal study illustrate an area of conceptual confusion in the theory. That is, to what extent can it be stated explicitly that exposure to uncontrollability causes anxiety, depression, hostility, negativity etc. Also, to what extent does one emotional state lead to another when exposure to uncontrollability is prolonged ? This issue is highlighted in Chapter 5 when the reliability of using psychophysiological variables as a means of assessing various emotional states is assessed. Bearing in mind these data, there have also been a number of criticisms levelled at the theory, both as a model of reactive depression and as an explanation of the behavioural findings from the human experiments. It is the latter criticisms that are of concern here, and the most detailed critique had been made by Costello (1978).

Costello examines six experiments and these will now be outlined.

1. The first study (Miller & Seligman, 1973) involved depressed and non-depressed subjects who rated their

certainty of success or failure on chance and skill tasks. Success and failure on each task was equiprobable, and it was found that non-depressed subjects showed greater changes on the skill task. Seligman claims that this was due to the depressed subjects having the perception of response outcome independence. Costello argues that if this was the case then the depressed subject should show no differential responding to success or failure. It is also unclear whether the effects were due to the perception of response outcome independence or simply due to lower motivation. It is also possible that the depressives had a more accurate perception of the experimental contingencies as these were fixed as being random by the experimenter.

2. Miller & Seligman (1975), employed a noise task followed by an anagram solving task and found deficits in those subjects who had been exposed to inescapable loud noise. Again Costello argues that these deficits are not necessarily a function of the perception of response outcome independence, but maybe due to a lack of interest.

3. Miller, Seligman & Kurlander (1975), used a line matching task and subjects had to rate their expectancies of success or failure. Depressed subjects showed lower expectancy changes after success compared to non-depressed subjects. But there were no differences after failure and also no differences between groups when subjects were asked whether success/failure was due to their own responding.

There were no differences either on a discrimination learning task or in the latency to turn off loud noise.

4. Miller & Seligman (1976), used a triadic design with estimations of success or failure. Costello notes that there were few significant results and all of these could be explained as being due to a lack of motivation. Also, even though the inescapable subjects did have the perception of response outcome independence, there were no differences between depressed and non-depressed subjects.

5. Klein & Seligman (1976), carried out two experiments using a triadic design with a pretreatment of escaping loud noise using a finger shuttle box. Again the deficits observed were not necessarily a function of the perception of response outcome independence.

6. Klein, Fencil-Morse & Seligman (1976). Depressed and non-depressed subjects were given solvable or unsolvable discrimination problems, or no problems at all, and tested on an anagram solving test task. Various levels of instructional set were employed and it was found that failure was not so important as the chance to externalise attributions. It seems that the depressives' poor performance was due to self blame rather than the perception of response outcome independence.

Costello (1978), lists a number of further objections:-

1. The cognitive deficit is postulated as being due to the perception of response outcome independence but there is no clear cut evidence that there are group differences on this.

2. Most of the data can be interpreted as due to poorer motivation hence the loss of reinforcer effectiveness hypothesis (Costello, 1972) is probably as adequate as the learned helplessness theory.

3. Uncontrollable loud noise could be considered as an extinction procedure, so that any deficits observed could be due to a loss of reinforcement.

4. Helplessness may not be the only or necessary response to the perception of response outcome independence. Other alternatives could be anger, puzzlement, or disinterest.

5. Another problem relates to the use of yoked controls. If a subject who is insensitive to noise is yoked to a subject who is sensitive to noise then the deficits observed would be due to differential sensitivity to noise and not due to differences in control.

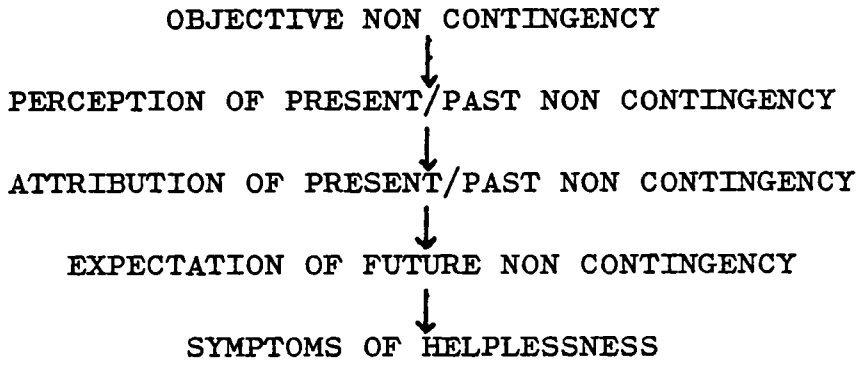
6. The use of the Beck Depression Inventory is also rather dubious as it has a section in it that relates to endogeneous depression which would not be applicable to learned helplessness depressives.

7. There is very little evidence that exposure to uncontrollability causes mood changes. For example, Klein et al (1976), did not report these data as "the measures were crude". Miller & Seligman (1975), found no main effects or interactions.

Seligman (1978), replies to Costello and makes the following points. Firstly, the criticisms of the experimental data are not necessarily valid. Miller & Seligman (1975), have shown mood changes which reach statistical significance. The Miller, Kurlander & Seligman (1975) study found that even though only one result reached statistical significance the other results were in the right direction. In the Miller & Seligman (1976) study, subjects were asked about their motivation and there were no differences between groups. Costello's (1972) alternative theory of reinforcer ineffectiveness is inadequate as subjects do not report it, they also behave as if they cared. Reinforcer ineffectiveness does not predict cognitive deficits neither does it predict increased anxiety and hostility. There are no self report data that suggest that the deficits are due to anger, disinterest, or puzzlement. The criticism about the use of yoked controls is invalid as it is equally possible that it is the noise sensitive subject who is in the escape group. If subjects are randomly assigned to groups then any effects due to differential sensitivity will be cancelled out anyway. As cognition is an inferred construct, it can only be measured indirectly. The support for this construct is that subjects self report the perception of response outcome independence and also subjects who have been exposed to the helplessness pretreatment have difficulty in perceiving that control is now available to them in the test phases of the experiment.

Apart from the studies that have yielded results that are contrary to the predictions from the theory of learned

helplessness, and also the criticisms that have been proposed by Costello (1978), the theory as it stands has a serious shortcoming as far as the data from the human experiments are concerned. The increased behavioural repertoire of the human subject and the increased capacity to represent environmental events requires that the theory operates at a level that cannot deal with the subjective structuring and interpretation of external events. The theory as it stands cannot deal with such factors as the context within which the uncontrollable event is experienced, or the importance of the outcome. It is not just the perception of response outcome independence but also the cognitive processes that are needed to generate expectancies about the future. The model underlying helplessness has been modified:-



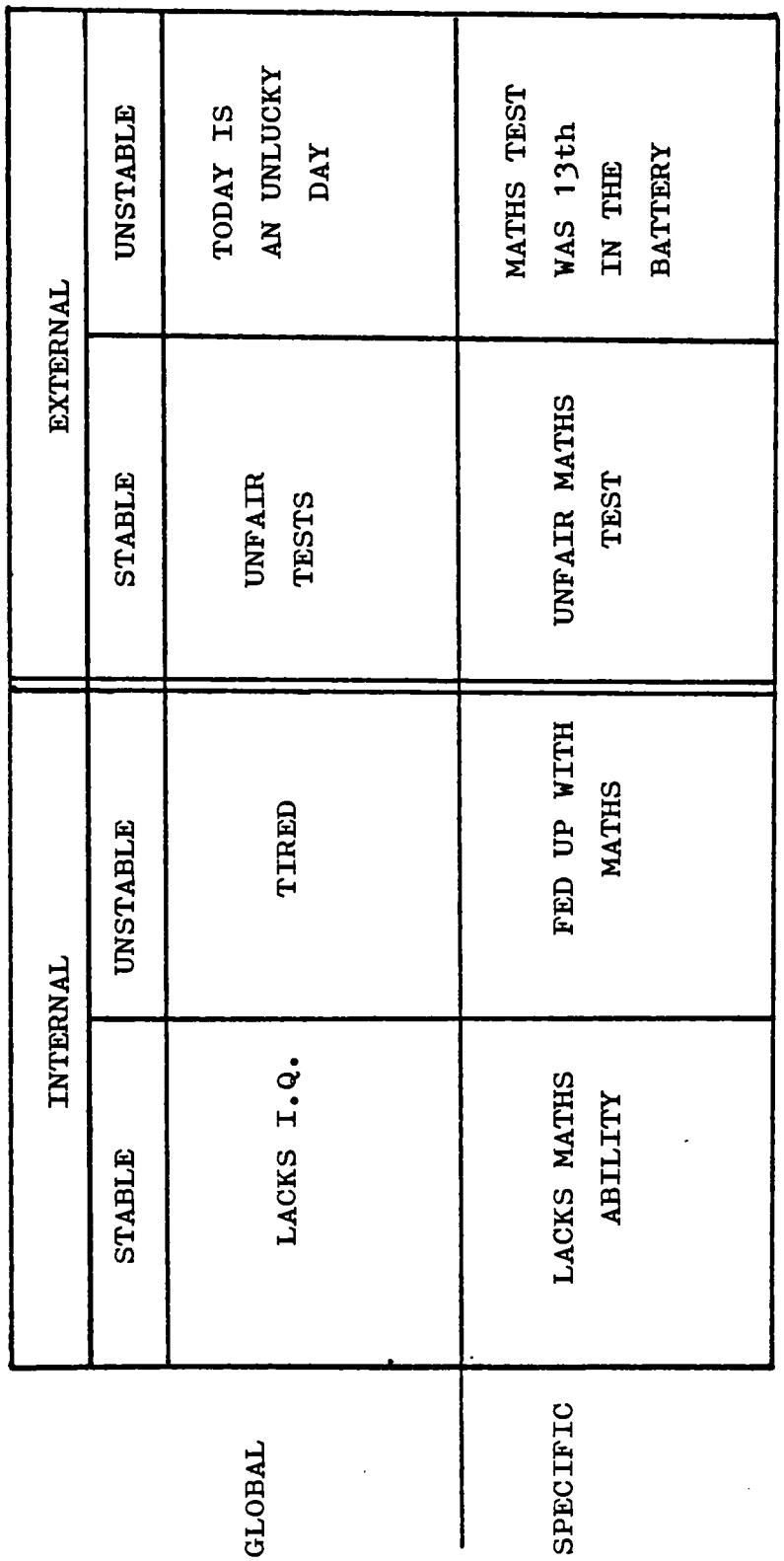
The main differences between the original formulation of the theory and the reformulation lies in the middle steps of the diagram. Both models postulate that the environmental non contingency must be perceived by the organism in order to be effective, but the reformulation states that an appropriate attribution must be formed about the non contingency. Given that this occurs and an expectancy of future non contingency results then the

subject will display symptoms of helplessness. The sttributional components have been formulated in some detail by Abramson et al, 1978, and these are based on Weiner's attributional model (1972, 1974), of:-

(internal/external) X (stable/unstable)

but with the added dimension of (global/specific). These dimensions form an attributional matrix which may be illustrated as follows. A student who fails a maths test (an event which is beyond his control), can form the following attributions about his failure:-

FIGURE 3. ATTRIBUTIONAL COMPONENTS OF HELPLESSNESS



Seligman (1978), maintains that this attributional matrix is orthogonal to the underlying dimension of uncontrollability, and as such the subjective attributions will determine the way in the perceived non-contingency will be expressed as helpless behaviour. If a student forms an internal, stable, and global attribution about his failure on a maths test, for example, that he lacks I.Q. then this will maximise the likelihood of deficits which will generalize and endure. The dimension of internal/external relates to individual differences in the way in which people reach conclusions about the cause of the uncontrollability. For example, one subject may conclude that the task is impossible to do (universal helplessness) and another may conclude that he does not have the ability to do the task (personal helplessness). Universal helplessness is consistent with the old helplessness hypothesis whilst personal helplessness is a refinement as it is not a clear case of uncontrollability as the subject believes that there are responses in the repertoire of others that could be effective.

Abramson et al. (1978) have extended this to distinguish between notions of failure and uncontrollability. Tennen (note 3 in the Abramson paper) has suggested that the term uncontrollability should be abandoned altogether and that the term failure should replace it as most human experiments involve experimenter induced failure and quote the term failure in preference to the term uncontrollability. Weiner (1972), proposes that success/failure refer to outcomes. Success refers to obtaining a desired outcome and failure refers to not obtaining a desired outcome. Abramson et al

(1980) suggest that failure is a subset of uncontrollability involving bad outcomes. As both good and bad outcomes occurring independently of responding lead to helplessness deficits, failure is a part of helplessness. Whether failure can be neatly subsumed under the rubric of helplessness is a point which will be discussed later. It will be argued that the human experimental evidence supportive of learned helplessness largely involves tasks where the subjects have been trying to exert control, are unsuccessful, and receive bad outcomes. It may be the case that failure is a qualitatively different experience to helplessness but also leads to similar deficits - a situation which is not entirely dissimilar to the controversy of associative vs. motor activity deficits in the animal literature.

A related consideration is the role of task importance, that is, the significance of obtaining or not obtaining the desired outcome. It is intuitively obvious that the importance of the task or event will determine, at least in part, the behavioural effects that will occur when the subject is unable to exert control over the event. The theory of learned helplessness stresses that it is the underlying dimension of controllability that is of paramount importance and yet the nature of the event is also crucial. For example, the perception of non-control over the sunrise is unlikely in helpless behaviour. Similarly, winning a large sum of money (uncontrollable as opposed to earning the money which would be controllable) may cause a behavioural deficit (not going out to work), but does not mean to say that other characteristics of helplessness will be found e.g. depression.

The original theory of helplessness (Seligman, 1975) had two inadequacies (Abramson et al., 1980) which are resolved by the reformulation. These inadequacies relate to the fact that whilst uncontrollability causes deficits in human subjects, these subjects will attempt to decide what the cause of the uncontrollability is. That is, objective noncontingency is predicted to lead to helplessness deficits only if the expectation of non-contingency is present. The old model did not specify under what conditions the perception that responses and outcomes are independent in the past or present will lead to the expectation of non-contingency in the future. The reformulation suggests that it is the attribution that individuals make at the present that is an important determinant of their subsequent expectations for future non-contingency. These expectations determine both qualitatively and quantitatively the expression of helpless behaviour.

The implications of the reformulation are as follows, an internal attribution which will result in personal helplessness as the attribution (that is, that the subject is unable to exert control but others can exert control), will lead to low self-esteem. The second implication is that stable attributions will lead to deficits which maintain for a longer period of time than deficits which occur with an unstable attribution. Thirdly, a global attribution will lead to greater generalization of deficits than a specific attribution. Another implication is that the severity of the deficits will be determined by the strength or certainty of the expectation of non-contingency (Abramson et al., 1980). The death of a loved one will be

associated with deficits of greater severity than failing a driving test due to the greater certainty of future non-contingency in the former situation.

Having described the reasons why the attributional reformulation was thought to be necessary, what it involves and its implications, it is necessary to consider the validity of such a model and to evaluate the experimental evidence behind this model. Firstly, Abramson et al. (1980), point out that both attributions and expectations are hypothetical constructs i.e. they are hypothesised events mediating between objective non-contingency and subsequent helplessness performance deficits. This means that there should be other ways to assess attribution and expectancy beyond the use of verbal self-report. The potential for developing an adequate technology for differentially evaluating attributions is something to be pursued.

Abramson et al. (1980) describe three classes of evidence which are relevant to the validity of the attributional reformulation. These will be considered in turn.

1) DEFICITS PRODUCED BY LEARNED HELPLESSNESS

Whilst both the old and the new helplessness theories explain the deficits resulting from exposure to uncontrollability, but the reformulation explains why the expectation transfers to new situations and how the time course of the deficits is determined. Results from previous human experiments may be made more understandable in the light of the reformulation. Roth and Kubal (1975) tested subjects who were led to believe that the task was either

important or unimportant. Helplessness deficits were observed only with the former group. The original helplessness theory would predict deficits in both groups, but this finding may be explained post hoc in the light of the reformulation. That is, that subjects who believed that the task was important probably formed global, internal, and stable attributions which maximises the certainty of the expectation of future non-contingency - hence producing deficits.

Similar explanations hold for considering the efficacy of therapy and immunisation, for example, therapeutic intervention which shifts a global attribution to a more specific attribution will lessen the extent to which the deficits will generalise. This also explains how debriefing the subject after the experiment ensures that helplessness is not taken out from the laboratory as the debriefing minimises the certainty of expectation for future non-contingency. Studies which have found facilitatory effects after uncontrollable pretreatments may be explained in terms of internal, specific, unstable attributions which lead the subject to compensate for their previous failure.

2) ATTRIBUTIONAL EVIDENCE

Four studies are relevant here. Klein et al. (1976) found that non-depressed subjects with an uncontrollable pretreatment performed poorly irrespective of whether they attributed their helplessness to internal or external factors. Tennen and Eller (1977) found that failure on easy problems (internal, specific, global attribution) produced deficits whilst failure on difficult problems (external, stable, specific) did not. Abramson et al. (1980) argue that the task difficult condition did not produce deficits due to the specificity of the attribution. Hanusa and Schultz (1977) and Wortman et al. (1976) found no performance deficits with either internal or external attributions. The fact that no basic helpless deficits were observed limits the value of interpreting these studies. Finally, Dweck (1975) has demonstrated that attributional retraining (i.e. to lack of effort to lack of ability) enables schoolchildren to do better.

3) EXPECTANCY CHANGES IN SKILLS TASKS

These changes will be determined in part by the stability attribution and are not necessarily a direct measure of a central helplessness deficit. The finding that helpless and depressed individuals show similar expectancy changes is supportive to a helplessness model of depression but not to helplessness per se.

So, as Abramson et al. (1980) have indicated there are supportive studies to the reformulation but the majority of these data are a re-examination of previous studies from the viewpoint of the reformulation. There are still relatively few studies that directly test the predictions from the

reformulation. Despite the reformulation there is still a controversy about the underlying mechanism to the deficits and also with respect to the concept of failure. It is also a significant finding that there are a number of studies which have failed to demonstrate helplessness deficits at all.

Confer (1978) found that the essential feature is that the subject holds the expectancy that the particular situation is potentially under their control. This illustrates the difference between expectancy and the perception of response outcome independence. Other studies have investigated the attributions which bridge perceptions and expectations in the manner proposed by Abramson et al. (1980). Campbell (1979) observed that subjects who made global attributions did worse than subjects who made specific attributions, but the actual helplessness deficits were non-significant. Pasahow (1980) similarly found that global attributions led to greater generalisation of the helplessness deficits. A slightly different type of evidence using attributional re-training to alleviate helplessness deficits. That is, to attempt to change the existing attribution (e.g. global) to a different attribution (e.g. specific), which in this case would limit the generalization of the deficits. Sobelman (1979) claims that this was effective in males but not in females, but did not establish a significant helplessness deficit in the first place. Raps et al. (1980) have found that affective states can be modified by manipulating types of thoughts (i.e. pleasant or unpleasant), so it is clear that cognitive process does affect mood. Whilst there are these studies which are supportive to an attributional approach, they fail to provide definitive tests

of the model. As Abramson et al. (1980) point out more research needs to be done and the evidence which they cite is often a post hoc explanation of experiments (in terms of attribution) that did not fit into the original helplessness model. The fundamental questions of whether subjects do form attributions and whether these attributions are significant determinants of behaviour have not been answered satisfactorily. The suggestion is that this issue needs to be verified and the situational circumstances that lead to the formation of attributions need to be delineated clearly. This extension into attribution theory has been a fairly central pathway of research in recent years, however, there are also peripheral avenues that have been explored which focus on the original helplessness model. For example, Kacher (1977) suggested that the deficits were not due to the perception of response outcome independence but due to the subject rejecting simple hypotheses and testing complex hypotheses. He was able to establish that this was not the case. Cole and Coyne (1977) found that subjects exposed to failure induction do not report feelings of personal helplessness. They suggest that rather than the perception of response outcome independence, their deliberation on the impossibility of the pretreatment interferes with the anagram task. Buchwald and Coyne (1978) further suggest that learned helplessness has been applied to any impaired performance and that in the learned helplessness literature uncontrollability is synonymous with experimenter induced failure. Coyne et al. (1980) report findings that learned helplessness only occurs if the subject is given an appropriate rationale. Their subjects became anxious and

hostile rather than depressed. The rationale given to the subjects was critical and suggested to them that learned helplessness were successful if experimenter induced failure occurred, the presence of uncontrollability was insufficient.

To summarise, the theory of learned helplessness has considerable support from the experiments on human subjects. However, there are important fundamental issues that remain to be resolved.

There have been a number of studies that have failed to replicate the learned helplessness effect. Whilst some of these have been explained in terms of attributional theory, the number of unpublished theses reporting non-significant results using the standard helplessness design is not to be ignored.

The reformulation of the theory in terms of attribution theory has resolved problems of chronicity, generalization, and emphasises the role of the subject in the interpretation of the uncontrollable outcome. Whilst there are supportive data to the attributional dimensions used in the reformulation there is still doubt whether these dimensions are necessary and/or sufficient.

Finally, failure has been subsumed under the attributional framework which in terms of the reformulation renders it a component (under certain conditions) of helplessness rather than an alternative explanation to helplessness. There are data to suggest that helplessness deficits occur only when the experimental pretreatment involves experimenter induced failure. This issue will be considered in more detail later as it forms the basis of the second experiment to be reported in this thesis.

CHAPTER 4

OVERVIEW

On the basis of the preceding chapters it is necessary to outline the central issues of the theory of learned helplessness and furthermore to provide the context within which the experimental studies to be reported are embedded. The evidence so far provides clear support to the theory in that exposure to uncontrollable stimulation leads to interference on a subsequent learning task. That is, the contingency of response outcome independence is actively learned with the observable consequence of disrupting the learning of other response outcome contingencies. Seligman (1975), has proposed that such exposure to the dimension of uncontrollability is also characterised by other deficits and these are:-

a) motivational

b) emotional

c) cognitive

Whilst it can be seen that the subject who has been exposed to uncontrollable stimulation shows impaired performance, the justification for this classification is not clearly stated in the literature. First of all, the evidence suggests that motivational deficits do occur. This is shown by the passivity observed in the animal experiments and the longer latency to solve the problems in the human experiments. But it is not clear whether this motivational deficit is due to the perception of response outcome independence or whether it is due to a more generalised conditioned response suppression. Whilst the competing motor response hypothesis has been ruled out as an alternative explanation for shuttle box deficits, this does not preclude a more basic explanation in terms of the inhibition of response initiation.

It could be argued that this inhibition and the perception of response outcome independence are synonymous, but the underlying mechanisms differ. In the latter case the environmental conditions cause a psychological state of which a motivational deficit is a symptom. That is, there is necessarily a cognitive mediator. The latter case excludes the cognitive component and implies that the motivational deficit is a direct function of ineffective response initiation. The role of cognitive mediation is a central and an important issue that will be expanded.

Maier & Seligman (1976), suggest that the measure of conditional probability of solution and trials to criterion reflect the cognitive deficit on the anagram solving task. That is, it reflects the extent to which subjects are unable to perceive that the situation is now controllable. These scores are supposedly distinct from the failure scores and the latency score which measure the extent to which motivational deficits are evident. It can be seen that all the anagram scores correlate highly (Tables 3 and 16) and this indicates that the scores are measures of the same thing, i.e. performance on the anagram solving task. Thus the distinction between cognitive and motivational deficits lacks empirical validation and the justification is purely intuitive.

To summarise the argument so far, whilst deficits are observed the independence of cognitive and motivational deficits remains to be shown and the conceptual status of cognitive mediation is unclear.

Several conceptual and methodological problems arise when the emotional deficits are considered. Firstly, it is probably necessary to talk of a generalised emotionality in animals as fine discriminants of emotional state are not in evidence. Archer (1973), has found that such measures of defaecation, ambulation, inhibition of feeding, etc. which have been used as indices of emotionality do not correlate highly. The data collected by Weiss (1971a, b, c), clearly show a higher incidence of gastric ulceration in rats exposed to uncontrollable shock compared to animals that had been exposed to controllable shock. The differences that preclude direct comparison of the Weiss data with Seligman's data similarly do not allow the assumption that such ulceration occurred in Seligman's rats that had experienced uncontrolled shock. The observations of distress in the experimental animals seem to be the major evidence for emotional deficits in the animal helplessness literature.

The availability of self report data provides greater justification for the concept of emotionality in the human experiments. However, there are several problems concerned with the concepts of emotion and mood which need to be considered before investigating emotional deficits in helplessness. It has been proposed that somatic changes were the precursors of emotional experience (James, 1884; Lange, 1885) and more recently Cannon (1920, 1931) has suggested that the subjective component of emotion is secondary to the somatic changes. However, it has been shown that cognitive factors are important determinants of the emotional response (Lazarus, 1966; Schachter & Singer,

1962), in that the choice of an emotional descriptor is determined primarily by cognitive appraisal that reflects the individual's perception of the situation. Emotional changes have also been shown where an individual has been presented with non-veridical feedback of somatic events (Valins, 1970). So, an emotion is an interaction between somatic changes and cognitive appraisal of both interoceptive stimuli and environmental conditions, and as such is a complex response system. This is reflected in the finding that considerable autonomic change can occur without an equivalent change in self reported affect. The converse, that is a self reported affect change without equivalent autonomic change, can also occur. The emotional response system consists of two components, somatic changes and cognitive appraisal that do not necessarily respond congruently. Given this oversimplified account of the basic system, the further problem of emotional discrimination arises when mood affect check lists are administered to assess emotional changes. Firstly, when the subject is confronted with a list of 16 mood dimensions to check, to what extent can the subject discriminate 16 distinct mood states subjectively ? For example, do subjects discriminate between anxiety and depression ? It is possible that subjects label both of these dimensions as being negative and so shift towards these ends of the relevant continuum after helplessness training in response to an evaluative judgement of the task demands and their evaluation of the experimenter's expectancies, rather than in response to an evaluation of their own subjective affective state. Factor analytic studies of mood affect check lists typically show that items correspond to a few underlying factors. For

example, Bond & Lader (1974), found that the 16 items yielded three major factors. This suggests that of the many possible adjectives used to describe emotional state, the underlying emotion is of a far more basic nature. So, there are problems of definition, problems concerning the congruence of self report and somatic components, and problems concerning task demand factors and response sets. These are basic to any research involving emotion. There are problems which are more specific to the research on learned helplessness.

Seligman (1975), has suggested that exposure to uncontrollability causes anxiety and when the exposure is prolonged, depression results. There is a large literature which is also inconsistent concerning the psychophysiological concomitants of these mood states and also that exposure to uncontrollability results in mood changes and somatic changes.

Szpiler & Epstein (1976), conducted a study in which subjects were told that they might receive electric shock following countdown. One group was told that shock could be avoided by tapping rapidly during the countdown, and a yoked group that tapped rapidly was told shock would be random. The avoidance group expressed lower anticipatory anxiety and gave fewer anticipatory SCRs, (a measure often used as an index of anxiety, (Miller, 1979)). Other studies have found similar results (Bowers, 1968; Houston, 1972). However some studies have found no differences in anxiety ratings (Averill & Rosenn, 1972) and no differences in non-specific SCRs (Glass et al, 1973). There is supportive evidence to Seligman's assertion that uncontrol-

lability causes anxiety, but there are two issues that arise from this. Firstly, studies typically do not report changes in other mood dimensions such as hostility, aggression, etc. and so it is not known whether such changes do not occur or whether they were not considered to be important. Secondly, as mentioned before, it is not clear that such self reports are a function of a genuine identifiable subjective mood change or an artifact of task demand and the subject's evaluation of the experimenter's expectancies. There is little evidence that depression results from continued exposure to uncontrollable stimulation apart from the anecdotal evidence cited by Seligman (1975). It is highly likely that changes on the depression scale do occur but it is not clear whether such changes follow greater anxiety or whether changes in both the anxiety and depression scales occur contemporaneously. The fact that such changes occur over the course of a single test session reflect that they are easily induced and the experimental evidence to be presented shows that these changes are easily dissipated. In addition to this, there are problems relating to the concept of stress and the discrimination of emotional states. Cannon (1936) considered that essentially the same physiological responses occurred in pain, fear, hunger and rage. Ax (1953) however, was able to discriminate fear and anger induced by staged situations by skin conductance, respiration rate, and muscle potentials. Attempts to investigate types of emotional response have been impeded by the difficulty in inducing emotional changes in controlled laboratory situations (Greenfield & Sternbach, 1972).

A more generalised problem is the discrimination of an emotional state against what might be termed 'activation'. For example, massive sympathetic nervous system responses can be obtained without accompanying reports of emotions or emotional behaviour. Wenger et al (1960) achieved such results by nor adrenaline infusion. The same results can occur by using simple physical exercises. Within the context of the learned helplessness experiments, the interpretation of physiological data must be done with care as physiological changes may be due to activation caused by attention, expectancies and effort to respond and may not be a reflection of stress symptoms.

A further factor is that of cognitive influences. A subject who is aware of his physiological changes may make an effort to relax in the belief that this will aid his performance. It would be expected that such efforts would also have physiological concomitants. To summarise these points, emotional measures have intrinsic definitional problems. There are data supportive to Seligman's theoretical predictions as regarding the effects of exposure to uncontrollability but,

a) there are non-supportive studies
b) self report data may be contaminated by task demands and experimenter effects
c) there are no clear psychophysiological discriminants of different mood states.

Together with the previous points, the assumption that there are three discrete deficits is debatable and there are problems concerning the adequacy of the dependent variables as measures of these deficits.

A second point relates to the concept of a state of 'helplessness' and the necessity to invoke a cognitive learning model. The central feature of helplessness theory is the perception of response outcome independence, which implies a cognitive representation of past environmental contingencies and the formation of expectations regarding the nature of future contingencies. Whilst this seems to be intuitively acceptable, the necessity of postulating 'helplessness' as a distinct psychological state and using cognitive terms is not beyond criticism. The use of such terms provides for a more elastic theory with relevant implications for every day life experiences and also case history data, but there are problems in stating explicitly what is meant by these terms. This reflects differing levels of explanation within the basic theory structure. These are:-

a) a learning theory explanation of the animal data
b) a cognitive learning theory explanation of the human data
c) a clinical case history explanation of the data relating to reactive depression.

The reformulation of the theory of learned helplessness incorporates all three levels of explanation, but this may be at the expense of scientific testability as well as providing an oversimplification of the diversity of the data. An example of this problem concerns the necessity for a cognitive representation of the uncontrollable contingency. It is unclear how it can be established that such a representation exists. If it implies that the contingency can be self reported then clearly this will

hold only for human subjects. A different explanation of the deficits found in the human experiments may be necessary rather than an elaborate extension of the existing theory. So, to what extent can these data be relied upon as accurate indications of the underlying process? It is quite possible that there are different processes underlying the animal experiments and the human experiments. As Coyne et al. (1980) have suggested the human experiments all seem to involve experimenter induced failure and it is possible that the human deficits are mediated by dissatisfaction or the perception of incompetence rather than the perception of response outcome independence. This would suggest that interference with an activity rather than the central disruption of that activity. That is, something which detracts from superior execution of that activity rather than an inability to do that activity. So, whilst the justification for the reformulation lies in the inadequacy of the original theory to account for situational influences on helplessness relating to specificity and chronicity of the state (Abramson et al., 1980) it is possible that the original model is an inappropriate one in any case to explain the basic human data.

It could be argued that the behavioural deficits observed in the animal experiments are largely a function of maladaptive learning contingent upon severe biological threat in a fairly basic system, i.e. the theory of learned helplessness. The deficits observed in the human subjects are a function of a far less severe kind of aversive stimulation and this may not operate at such a fundamental level as the theory of learned helplessness has been formulated.

This has two main implications:-

a) that the deficits in the human subjects are subject to state and trait components that affect the response to the experimental conditions e.g. attributional and personality factors. Such factors are minimised in the animal experiments by the use of laboratory animals of known pedigree and developmental history;

b) whilst deficits are observed in both animals and humans after exposure to uncontrollable stimulation the underlying process may not be the same.

Given the experimental evidence from the animal research, there is considerable support for the theory of learned helplessness, but the similarity between the experimental paradigms does not necessarily mean that the theory of learned helplessness must be invoked to account for deficits observed in the human experiments. It will be argued that learned helplessness is, in actual fact, the most appropriate explanation for the human data, but the increased behavioural repertoire of the organism must also be taken into account. The reformulation of the theory of learned helplessness has achieved this, but other possibilities remain. Far from investigating the experiments in order to identify causes for the deficits the reformulation imposes an all embracing attributional super structure onto the original theory. There are problems associated with self-report data, some of which are considered by Nisbett and Wilson (1977). For example, subjects are sometimes unaware of influential stimuli, unaware of having responded and even when subjects are aware of the stimulus response contingencies self-reports may be based on a priori, implicit, causal theories rather than true introspection. In the laboratory such data may also be contaminated by subjects'

expectancies concerning the expectancies of the experimenter. An alternative explanation is that the deficits are due to greater dissatisfaction, and general dysphoria contingent upon the perception of failure, which is implicit in the human experimental paradigm and will be the subject of one of the experimental studies.

Again this suggests a more peripheral interference rather than a central disruption. It is as if the failure of the subject may distract his attention from performing the task in a competent manner. It can be seen that this is a different state of affairs to the disruption of test task performance due to a centralised expectancy of response outcome independence. This interpretation of events is not entirely dissimilar from the reformulation in so far as there is some provision for the subjective interpretation of events. But, it does not assume that causal attribution is simply a superstructure to basic expectations of response outcome independence. It proposes that deficits on the test task may be features of the evaluation of performance by the subject, possibly in terms of distractability. That is, a subject who is thinking about reasons for failure, upset about failing, will not be devoting the whole of his attention to the test task and hence will not be performing at full capacity. This seemingly trivial alternative may account for performance decrements without the need to invoke the theory of learned helplessness. The differences between the animal studies and the human studies are apparent and direct comparison is difficult to effect.

The reformulation does not allow this to be tested, in fact, because failure is subsumed under helplessness with

appropriate attributional components. This distinction can be effected experimentally however by manipulating both controllability and experimental information designed to affect the subjects beliefs about success and failure independently of their actual control over the events. So long as the experimental variables are defined operationally in terms of the actual experiment, the distinction between failure and response outcome independence is valid experimentally.

As has been noted previously, there have been many unpublished studies which have failed to show helplessness deficits using the standard helplessness triadic design. Whilst these studies may have contained methodological flaws it is still a possibility that human helplessness is not so reliable a phenomenon as the published literature suggests.

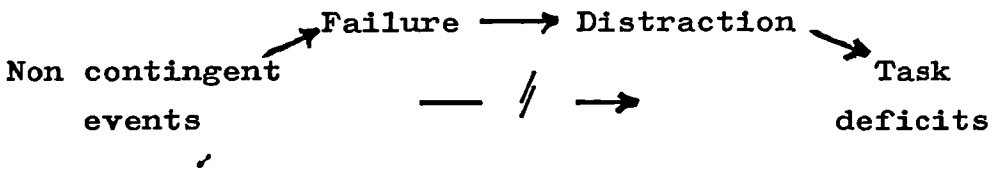
There have been studies that have failed to show deficits after uncontrollable pretreatments (Roth & Bootzin, 1974) and studies that have shown facilitation effects after such exposure (Roth & Kubal, 1975) which can be explained in terms of the reformulation. Whether the other unpublished non supportive studies can be accommodated quite so readily remains to be seen. In any case, such explanation is always after the fact and does not constitute an experimental test.

It is known that deficits can and do occur after exposure to uncontrollability but what does the subject perceive in such situations? That is, is response outcome independence actively learned leading to an expectancy of non contingency or does the experience of failure lead to distracting thoughts which interfere with efficient execution of the test task? These two processes are distinctly

different. In the first case the subject is directly influenced by uncontrollability.

Non contingent events → Expectation of non contingency → Task deficits

In the second case the performance of the subject is affected by intrusions which do not allow the subject to perform his best.



This is similar to a learning/performance distinction. The first case is where learning is being disrupted directly, the second case is where the expression of learning as performance is being interfered with. To summarise this point, does exposure to uncontrollable events directly disrupt later learning or does the failure experience lead to cognitions and thoughts that prevent the expression of learning as performance?

This is an important issue with respect to learned helplessness as it brings into question whether learned helplessness does mediate the task deficits at all. This will be the subject of the second experiment to be reported in this thesis.

The reformulation of the theory of learned helplessness may be depicted as in Figure 3. Presumably, one of the main reasons that a reformulation of the original theory was thought to be necessary was the experimental studies that failed to support the theory as it stood. The cognitive

representation is even more difficult to test in animals, and cognitive constructs (for example, preference and expectancy as used by Seligman and Johnston (1973) in their cognitive theory of avoidance learning) remain hypothetical constructs which are difficult to validate. There have been studies that have failed to show deficits after uncontrollable pretreatments, (Roth & Bootzin, 1974), and studies that have shown facilitation effects after such exposure (Roth & Kubal, 1975). The proposed curvilinear relationship between deficits and the amount of helplessness training has been superseded by the reformulation. Other contributory factors were the findings that the personality factor of External/ Internal Locus of Control and the state characteristics of attribution were also important determinants of exposure to uncontrollability. The reformulation takes account of these factors formally with the dimensions of global/specific, stable/unstable, and internal/external attributions previously described in Chapter 3. An alternative to this position dispenses with the underlying concept of uncontrollability and introduces the concept of failure. Whenever a subject is unable to control the stimuli in an experiment, does the subject perceive response outcome independence or does the subject perceive failure on a test ? This suggests that the deficits may be due to the dysphoria, discontent and lack of motivation and not a direct result of the learned dimension of response outcome independence. The deficits may even be an interaction between the perception of response outcome independence and also the perception of test failure.

It is necessary to ensure that perception of failure and the perception of response outcome independence are not

synonymous, and given the reformulation this is difficult to state explicitly. That is, given that a subject has been unable to control the stimulus in the experiment this will be experienced as failure to the extent to which the subject perceives that the task can be done successfully by other subjects. If the subject believes that no one can be successful, then he is unlikely to evaluate his performance in terms of failure, but in terms of the impossible demands of the experimenter. Contrariwise, if the subject believes that everyone else can do the test then he will interpret his performance as failure. The experimental design of the second experiment to be reported allows a test to evaluate whether subjects self report failure or lack of control and also to manipulate the information given to the subjects to attempt to assess the relative importance of these components.

It could be argued that this higher order concept of failure is superfluous as it is a function of lack of control with the appropriate attribution. The data so far do not suggest that this is the case, but there have been few direct tests of the reformulation of the theory. It could similarly be argued that the concept of control or the attributional components are superfluous as they are largely a function of a more basic conditioned inhibition of response initiation. It could also be the case that thoughts of failure distract the subject and so he/she does not demonstrate optimum performance on the test task. There are differences between human and animal experiments which do challenge the adoption of the helplessness model in the human experiments.

To summarise, there are several important and central issues yet to be resolved:-

1) are there three discrete deficits and to what extent are they independent ?

2) to what extent is a cognitive learning model necessary to explain the data ?

3) does the theory of learned helplessness need to be invoked and can it account for all the data adequately ?

CHAPTER 5

THE DEPENDENT VARIABLES

Seligman (1975), has proposed that learned helplessness is a general organismic debilitation characterised by deficits in cognition, motivation, and learning ability. Therefore research on learned helplessness requires that a variety of measures are taken - measures that relate to the experimental subject's feelings and cognitions, physiological reactivity, and also to the behavioural deficits associated with exposure to uncontrollable stimulation. This approach is also reflected in the literature dealing with research on emotion.

Lang's (1968) attempt to measure fear starts with the assumption that it is a response expressed in three main behavioural systems; verbal (cognitive), overt motor, and somatic. Lazarus (1968) takes a similar view - an emotion consists of motoric impulses, causal appraisal, and physiological changes. Such a classification in terms of three discrete components oversimplifies the behaviour and it is clear that these three systems interact to a considerable extent (Schachter, 1964). The picture is further complicated by the fact that these systems can function independently. A self reported state of anxiety may not necessarily be accompanied by the usual physiological changes associated with an anxious person. Similarly, all the physiological characteristics of anxiety may be observed and yet the person does not report feeling anxious at all. However, for the purpose of describing the dependent variables used in the experimental studies and deriving testable predictions it is a useful, if simple distinction to make.

This chapter will describe the dependent variables used in the experimental studies to be reported from the point of view of their relevance, measurement, and the analysis involved with these measures.

TEST DATA

The test data refer to the anagram solving task used to assess any behavioural deficits associated with exposure to uncontrollable stimulation, and also the Mill Hill I.Q. test.

Anagram Test

A series of twenty, five letter anagrams were used in the first study, as in the previous literature on helplessness studies (Hiroto & Seligman, 1975; Klein, Fencil-Morse, & Seligman, 1976). These were taken from a list prepared by Tresselt & Mayzner (1966), and could all be solved by the letter order 3-4-2-5-1. A subset of ten anagrams was selected for the main study.

Four major dependent measures were obtained for each subject on the anagram task:-

1. mean latency for solution.
2. the number of trials to criterion for discovering the solution pattern. The criterion was defined as the number of trials taken to include three successive trials with a response latency of less than 15 seconds each.
3. the number of failures to solve an anagram within 100 seconds.

4. the conditional probability of solving an anagram given that the prior anagram was solved.

Two measures of mean latency were calculated, one which omitted failures (i.e. mean solution time), and another which counted failures to solve as 100 seconds each. The main reason for using the anagram solving task was that it has been used extensively in learned helplessness experiments before, it affords several dependent measures, and it is a cognitive test in contrast to the instrumental pretreatment.

It was predicted that the differences in experimental conditions would be reflected in the measures on the test task. That is, the degree of failure/uncontrollability in the pretreatment phase would be reflected in the degree of deficiency shown on the anagram test task.

Non parametric and parametric analysis of variance was carried out on the raw data and the transformed data to assess whether there were any differences between groups. The major problem in using the anagram solving test lies in the fact that there are individual differences in anagram solving ability per se. Some control over this factor can be obtained from the verbal I.Q. score.

The Mill Hill Verbal I.Q. Test

The Mill Hill Test is a 34 item self administered test where subjects are required to select from a choice of six alternatives, the word which is closest in meaning to a given word. (The other half of the test, in which subjects are asked to supply their own definitions to given words, was not used). It is scored by summing the

number of correct answers, and after an age correction affords an index of verbal I.Q. To some extent the test measures something that is an important determinant of anagram solving ability, and in conjunction with self rating data on experience with crosswords/word puzzles etc. it allows the anagram scores to be weighted so that subjects should become equivalent. However, such weighting of scores is limited, as it depends on an adequate variance in the anagram scores that is also evident in the verbal I.Q. scores, and this covariation is not revealed in the data. These issues are dealt with more fully in the chapters on the experimental studies.

QUESTIONNAIRE DATA

The questionnaires used in the two experimental studies may be broadly classified as trait and state variables. Trait variables are those reliable, consistent underlying subject characteristics that can affect overall responding in the experiment. The relevant questionnaires are the Rotter Locus of Control Questionnaire, the Beck Depression Inventory, and the Eysenck Personality Questionnaire. State variables are those variables that vary over time, particularly during an experiment, and are typically check lists that relate to mood. Congruent with this, is that trait questionnaires are given once prior to the experimental session and the state questionnaires are given several times at appropriate points during the experiment. The distinction between trait and state is not clear cut - for example, scores on the Beck Depression Inventory will

change over time, particularly during the course of a depressive illness. Also, scores on the Rotter Locus of Control may change over time but the time course involved is such that it can be treated as a trait measure. The questionnaires will be considered in turn.

The Beck Depression Inventory

The Beck Depression Inventory consists of 21 symptom attitude categories. Each category describes a specific behavioural manifestation of depression and consists of a graded series of four to five self evaluative statements. The statements are ranked to reflect the range of severity of the symptom from neutral to maximum severity. Numerical values from 0 - 3 are assigned to each statement to indicate the degree of severity, and the total score is obtained by summing the scores of the individual symptom categories. The items were chosen on the basis of their relationship to the overt behavioural manifestations of depression and do not reflect any theory regarding the aetiology or the underlying psychological processes in depression (Beck, 1967). The inventory has been shown to be reliable (reliability coefficient of .86) using the split half method of reliability estimation. The inventory has been shown to be valid by relating highly to clinical ratings, predicting clinical change, and correlating highly with existing depression inventories (e.g. .4 - .6 with the Depression Adjective Check List, Lubin, 1965).

The Beck Depression Inventory (BDI) has been used to investigate the relationship between the learned helplessness deficits and reactive depression (Miller & Seligman, 1973). The inventory can be used to allow preselection.

Miller (1971), allocated subjects who scored 9 or above to depressed groups and a score of 8 or below to non-depressed groups. The prediction was that depressed subjects should be equivalent on the test task compared to non-depressed subjects who had undergone the pretreatment of uncontrollability. Depressed subjects who had undergone this pretreatment should show the most severe deficits of all. The scores on the Beck Depression Inventory can also be used to compute correlations between depression and measures of performance, ratings of perception of response outcome independence and mood/expectancy changes during the experiment (Miller & Seligman, 1973).

The BDI was used in the second experiment for two reasons. A preselection was not attempted, but administration of the BDI allows subjects who are depressed (a score of 9 or above, Miller, 1971), to be excluded from the study. The primary concern of the studies was to investigate components of the experimental paradigms and not to draw parallels between learned helplessness and depression, so it is reasonable to exclude depressed subjects (this was unnecessary as no subject scored 9 or above on the BDI). The BDI scores also afford an index of depressed mood that is an additional datum to be considered in the evaluation of the other dependent variables.

The appropriateness of the BDI is arguable however, as it is a tool constructed to measure depression in clinical populations. It is possible that it is too crude to distinguish between degrees of non-clinical depressive mood. Beck (1967), states that a 'moderately' depressed patient would score 25.4 (the mean). This is considerably

higher than the mean score of the mean score of the depressed subjects (13.75) reported by Miller & Seligman (1973). However, the BDI is quickly and easily administered and provides additional information about the experimental subjects.

In summary, the BDI is not directly relevant to the experimental studies as no preselection was involved. However, it allows depressed subjects to be excluded and is another source of mood data. It also allows the prediction that the BDI scores should correlate positively with deficits and mood changes associated with pretreatment of uncontrollability - i.e. the more depressed the subject to begin with the greater the susceptibility to perceive response outcome independence and the greater the potentiality to demonstrate deficits in the test task. Given the low variance of the BDI scores the testability of this prediction is questionnable.

The Rotter Locus of Control Questionnaire

The Rotter internal/external Locus of Control scale is a 23 item forced choice questionnaire with six filler items adapted from the 60 item James scale. It is scored in the external direction, that is, the higher the score the more external the individual. The scale measures the extent to which people believe that they are in control of life events. That is, an external believes as shown by the answers on the inventory that reinforcements occur in his life by chance or luck and are beyond his control. An internal believes that he controls his own reinforcers and that skill rather than chance causes achievement. The rationale is derived from social learning theory

(Rotter, 1966), that the potentiality of occurrence of a set of behaviours is a function of the expectancy of reinforcement and the strength or value of the reinforcement. Locus of Control relates to the expectancy of reinforcement which is an important feature in determining whether a subject is likely to become helpless or not as a result of exposure to uncontrollability. Exposure to uncontrollable stimulation is not sufficient to produce deficits, the important aspect is whether the subject actually perceives the contingencies to be beyond his/her control (Seligman, 1975). It is highly likely that subject characteristics that contribute to the formation of this perception are likely to contribute to a high score on the Locus of Control Inventory.

The questionnaire has extensively normative data (Lefcourt, 1976), but there are little data on the reliability or validity of the scale. Miller & Seligman (1973), cite a test-retest reliability of .76 over three months. The questionnaire has been used for preselection (Miller & Seligman, 1973), with varied results. This may be due to selecting above and below the mean rather than selecting extreme scores at either end. The data collected from the first study were inconsistent and this may be due to the heterogeneous subject sample. Although the subject sample in the second study was more homogeneous (on sex and age), the Rotter Inventory was administered to provide information about externality should the results show a similar inconsistency. It would be expected that externals are more likely to become helpless compared to internals as they attribute reinforcement to external causality

rather than personal control. However, "the strong situational determinants of the competitive laboratory task limits prediction" (Rotter, 1966).

The Eysenck Personality Questionnaire

The EPQ has an extensive history and literature, and this will be outlined briefly as the EPQ is not a major variable of interest in the experimental studies. The questionnaire consists of 90 items which yield the four dimensions of psychoticism (P), extraversion (E), neuroticism (N), and the lie scale (L). Experimental studies have shown that the N factor is closely related to the inherited degree of lability of the autonomic nervous system, whilst the E factor is closely related to the degree of excitation and inhibition prevalent in the central nervous system. The psychotism factor P, has been referred to as 'tough mindedness' and a high P scorer may be characterised as cruel, inhumane, lacking in feeling and empathy (Eysenck, 1970). The lie scale L attempts to measure a tendency by subjects to 'fake good' but it also seems to measure some stable personality factor which possibly relates to social naiveté. Test retest reliability is high (.8) and extensive normative data are available (Eysenck, 1970).

The EPQ was included in both studies to identify atypical personality profiles that may account for outlying or discrepant scores and to enable post-hoc correlations with the Rotter, Beck inventories and possibly with other dependent variables. It is difficult to formulate clear cut predictions from the EPQ although personality may be an important determinant of both physiological and

behavioural responding in the learned helplessness paradigm. As Seligman has proposed that the uncontrollability axis can be learned as any other in the operant training space, then helplessness may be influenced by conditionability and hence a function of the extraversion dimension (Eysenck, 1970).

The EPQ has not been used before in the learned helplessness literature and is used here as an exploratory variable, but it could be predicted that subjects scoring high on the extraversion factor are more susceptible to the perception of response outcome independence as they depend more on environmental stimulation.

The Mood Check Lists

Mood has been defined by Nowlis (1965), as "a multi-dimensional set of temporary reversible dispositions". Mood can change dramatically in short time periods and be accompanied by changes in physiology and performance. Hence, it is necessary to take mood states into account when testing human subjects, especially when mood changes are predicted as in the learned helplessness experiments.

Exposure to uncontrollable stimulation is more aversive than exposure to the same stimulation when it is controllable, and so mood changes relating to increased anxiety, activation, tension etc. and decreased relaxation, pleasantness, calmness are to be expected. This increased anxiety gives way to depressed affect and this complicates the measure, as changes predicted (such as increased deactivation, lethargy etc.) are contrary to the expected anxiety changes. Given that different experimental

conditions are contained within the study, overall predictions are that uncontrollability will be associated with increased negative mood changes and decreased positive mood changes compared to the other experimental conditions, also, that such mood changes should revert after the test task where all groups are exposed to identical conditions.

Mood may be inferred from overt behaviour or most usually in the laboratory by self report techniques. Because so little is known about mood there are at present few reliable measures other than those based on verbal items (Nowliss, 1965). Lazarus (1966), has noted three main problems in using the self report technique:-

1. individuals may use different labels for the same mood (or even the same person using different labels for the same mood on different occasions).
2. distortion due to 'faking good' where subjects wish to present themselves in a good light.
3. unconscious bias - that is, the subject sees himself in a light incongruent with reality.

Using averaged data reduces these biases but mood data are usually associated with a large amount of error variance. The experimental studies used adjective check lists, where the subject is presented with a list of adjectives and is required to indicate the extent to which the adjective applies to him at that time. The lists used were the Mood Adjective Check List (Nowlis & Green, 1957) in the first study, and the Mood Rating Scales (Bond & Lader, 1974) in the second study.

The Nowlis MACL

The bases of the MACL were the four intuitively derived dimensions of mood, namely:-

1. 'activation/deactivation',
2. 'positive and negative' social orientation,
3. 'control and lack or loss of control',
4. 'positive and negative' appraisal.

Nowlis & Green (1957) selected 120 adjectives representative of these dimensions and gave the resulting check list to 450 male students in a variety of mood inducing situations. Factor analysis revealed 12 factors which were unipolar contrasted to the expected 4 bipolar factors.

The check list used in the first study was based on the original check list (Nowlis & Green, 1957), and consists of 45 adjectives. The subject is required to check each adjective as to whether it definitely applies, applies slightly, they are undecided, or does not apply to their mood at that moment. These adjectives are then scored and summed into the twelve categories previously found namely:- aggression, anxiety, social affection, concentration, nonchalance, depression, pleasantness, activation, deactivation, startled, sceptical, and egotism. There are several problems associated with this scale - for example - it is usual to score the list from 4 (definitely applies), to 0 (does not apply) for each adjective, so giving a score of 1 to the category 'cannot decide', which is questionnable. There are also problems with the forced compliance nature of the list. That is, every adjective has to be checked and there are only four

discrete checks available. However, these problems are minimized when the mood data are compacted into the mood dimensions as the variance for the mood scales increases.

The Lader ACL

This check list has the advantage of using a continuous analog scale (Bond & Lader, 1974), as opposed to discrete checks. This means that the list is easier to use, allows finer discrimination, and also reduces the difficulties of response sets and the artificial distribution of positive and negative responses (Joyce, 1968). The questionnaire consists of sixteen 100 mm scales. The scales are scored by measuring in mm's from the end of the line to the subject's mark. The questionnaire has face validity and unknown reliability as the scales are sensitive to state mood changes. The actual scales are given in the appendix and can be factored into dimensions of:-

1. intellectual impairment
2. bodily impairment
3. calming effects
4. others (Norris, 1971).

Bond & Lader (1974) extracted three factors, which are:-

1. alertness
2. contentness
3. calmness.

This check list has been used to assess mood changes in drug trials with effectiveness and was used in the second study mainly to allow finer discrimination of mood states

as it used continuous scales.

Analysis of mood data is a problem as there are individual differences in the way that subjects use the scales. Analysis of variance can be used on the change scores but these are likely to be a function of the pre-stimulus level, (Mackay, 1980). Statistical treatment of the raw scores using the autonomic lability scale of Lacey (1956), or analysis of covariance may be appropriate if this is the case.

However, the easiest way to appraise the mood data given the sources of inconsistency is by use of graphs together with tables of means and standard deviations. Analysis of variance was also carried out on the raw data and on the change scores.

The Post Experimental Questionnaire

These questionnaires differ according to the experimental condition to which a subject has been assigned and also differ across the two studies. But, in general the questionnaires are given at the completion of all tests and are largely ratings of the stimuli, motivation, attributions of success and failure and reports on the subject's beliefs and expectations during the experiments. This is important firstly to check whether the subject understood and believed the contingencies to which he was exposed, (particularly necessary when non-veridical feedback is used) and secondly, to gain information about subjective stress and other factors that may contribute to the responding of the subject.

PHYSIOLOGICAL VARIABLES

> "Whilst one would hesitate to claim that physiology alone can define 'so called mental mechanisms' or 'reveal' the 'phenomena of mind' as Darrow has claimed, the appraisal of physiological responses as an aspect of a total, complicated behavioural response pattern increases our understanding of an organism's behaviour in an experimental situation."
>
> Martin, 1973.

The measures taken in the first study were skin resistance and heart rate. The main study used these two measures and also the measure of respiration. The evidence suggests that exposure to uncontrollable aversive stimulation is more noxious than exposure to controllable aversive stimulation. This should be reflected in the physiological activity of the organism but the literature fails to show consistent findings. This is a function of three influences - namely:-

1. the equivalence of the paradigms across the experiments.
2. the equivalence of the experimental conditions within an experiment, and across subjects.
3. the complexity of the physiological processes that are being recorded.

The measures used are usually measures of autonomic reactivity, and the autonomic nervous system is largely in a state of homeostasis contingent upon the interaction of sympathetic and parasympathetic control. The sympathetic nervous system (SNS) in general serves to provide a 'fight or flight' reaction, so that anxiety may be described physiologically as an increase in heart rate, an increase in skin conductance, and a faster breathing rate. However, this is usually against the antagonistic action of the

parasympathetic nervous system (PNS), so that these physiological changes are only the apparent effects of SNS activity as the real cause is a complex interaction of both SNS and PNS control (Sternbach, 1966). With this complexity in mind and the findings that physiological changes are not necessarily always associated with cognitive or behavioural changes, care must be taken in evaluating physiological data. Each measure will now be considered in turn.

Skin Resistance

There are various direct methods of estimating sweating e.g. direct count of active sweat glands (Ferreira & Winter, 1963), thermal conductivity (Adams & Vaughn, 1965), but electrical methods are favoured because they tend to be more accurate, and are easier to monitor in an experiment. Electrical methods may be either exosomatic - where a small current is passed between two sites and the resistance is measured (Féré, 1888), or endosomatic - where the naturally occurring potential difference between two skin sites is measured (Tarchanoff, 1890). Exosomatic recordings tend to be the most used in the literature.

There are two forms of sweat glands found on the human body; these are denoted as apocrine and eccrine (Schiefferdecker, 1917, 1922). Apocrine glands are of little importance in the production of electrodermal activity. Eccrine sweat glands have the property of responding to both thermal and signal stimuli, but activity due to thermoregulatory control is normally seen only when the ambient temperature exceeds 30°C. Darrow (1933), has

suggested that the role of eccrine sweating on the palms is more concerned with grasping behaviour than with evaporative cooling.

There are two possible mechanisms for the physiological basis of the skin resistance level (SRL):-

1. that changes in vascular tone underlay the SRL.
2. that sweat gland activity causes the SRL.

Experimental studies using local perfusion of atropine (which abolished the skin resistance response but had no effect on vascular activity), and bretyllium (which abolished vasomotor changes without affecting the SRR), lead to the conclusion that the SRR is entirely dependent on sweat gland activity (Dale & Feldberg, 1934; Lader, 1970). It is worth mentioning at this point that sweat glands are exceptional as they are innervated solely by SNS postganglionic fibres and yet the transmitter substance is acetylcholine. So sweat glands do not participate in the mass innervation of SNS activity caused by adrenal medullary secretions and hence one cannot generalise from sweat gland responding to activity in the rest of the SNS.

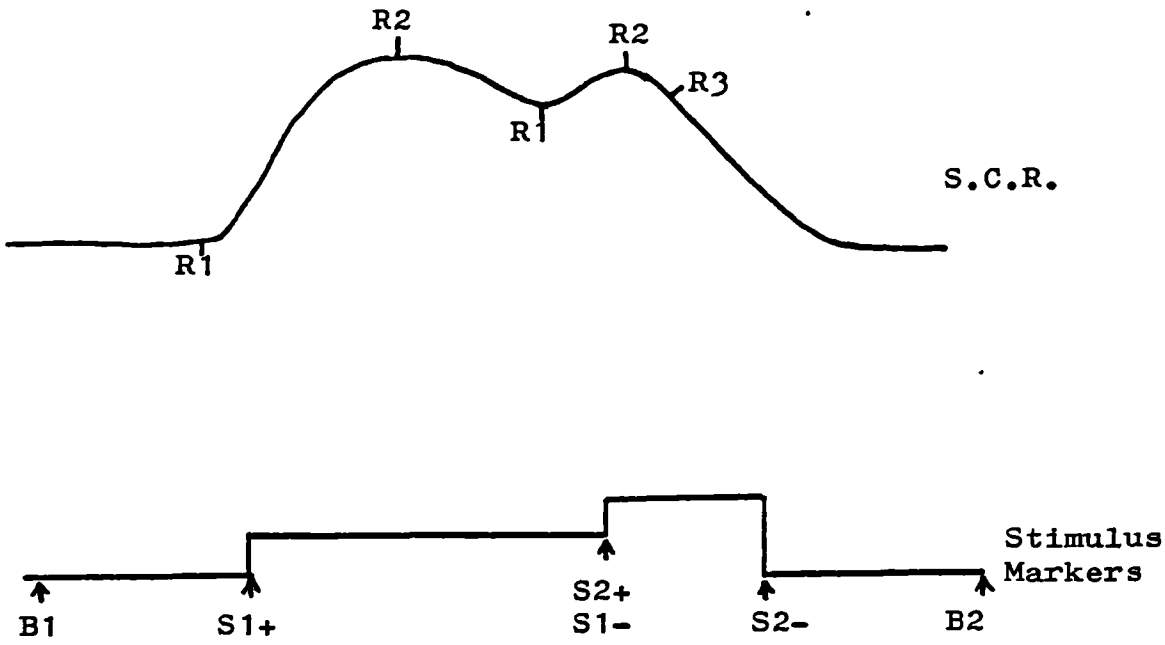
The discharge of sweat at the skin surface is in part a product of pulsatile contractions of a myoepithelial spiral chain surrounding the sweat duct, which also favours ductal reabsorbtion (Nicolaidis & Sivadjian, 1972). It is thought that this innervation of the myoepithelial chain may be adrenergic (Goodall, 1970).

A brief mention of the terminology is necessary. The terms PGR (psychogalvanic response), GSR (galvanic skin response), SRR (skin resistance response) are all

equivalent, but SRR is more commonly used. There is a distinction to be made between SRR and SRL (skin resistance level). The skin resistance level is a basal level of the subject (tonic), against which skin resistance responses (phasic) are measured. However, for statistical reasons (i.e. the distribution of skin resistance measures is often skewed), and more importantly for biological reasons, skin resistance is usually expressed as conductance (hence SCL - skin conductance level, SCR - skin conductance response). The biological basis for this is as follows. Ohm's law states that V=IR, where R is the resistance, I is the current, and V is the voltage. If I is kept constant, the voltage (V) recorded is directly and linearly related to the resistance. This forms the basis of the constant current method for measuring skin resistance. Similarly, as I=V/R, conductance may be measured by a constant voltage method. Darrow (1934, 1964), found that the rate of sweat secretion is linearly related to skin conductance. Also, Thomas & Korr (1957), found that conductance varies linearly with the number of active sweat glands. Since sweat glands may be conceptualised as low resistance pathways through the stratum corneum of the skin, they can also be represented electrically as a number of switchable resistors in parallel with each other and the high resistance of the stratum corneum only in terms of conductance since resistors in parallel are not additive like resistors in series.

Thus the recorded conductance is G1=GO+NG, where GO is the conductance of the stratum corneum, N is the number of active sweat glands and G is the mean conductance of an active sweat gland. As GO is negligible (about 1 mho

FIGURE 4. SKIN RESISTANCE MEASURES



STIMULUS NOTATION

B1 :- RESISTANCE LEVEL AT ONSET OF TRIAL

S1+ :- RESISTANCE LEVEL AT ONSET OF NOISE

S1- :- RESISTANCE LEVEL AT OFFSET OF NOISE

S2+ :- RESISTANCE LEVEL AT ONSET OF FEEDBACK SLIDE

S2- :- RESISTANCE LEVEL AT OFFSET OF FEEDBACK SLIDE

B2 :- RESISTANCE LEVEL AT OFFSET OF TRIAL

RESPONSE NOTATION

R1 :- RESISTANCE LEVEL AT ONSET OF RESPONSE

R2 :- RESISTANCE LEVEL AT PEAK OF RESPONSE

R3 :- RESISTANCE LEVEL AT 50% RECOVERY OF RESPONSE

N.B. Both response level and event time are given for each data point allowing derivation of response latency, rise time etc.

cm^2 of skin) G1 is proportional to N. If a stimulus is applied, G1 increases to G2 as N1 increases to N2. The number of additionally active sweat glands whose activity constitutes the SRR is given by N1-N2, which is proportional to G1-G2. Changes in resistance bear little relation to changes in sweat gland activity (Lader, 1970). The atropine perfusion study supports this as skin conductance drops steadily as a regular monotonic decrease. Skin resistance has erratic changes in size before an abrupt drop to zero.

So, measurement of skin resistance is usually taken and the resultant scores are converted to conductance. Typically the distribution of the skin conductance data necessitates a log transformation prior to analysis.

An idealised resistance recording is shown in Figure 4, together with the commonly used measures taken from the recording. In conjunction with the time based stimulus marker channel, various derived measures (e.g. rise time, recovery time), can be calculated. As there are minor differences in measures and analysis the details are presented in the relevant experimental studies. Previous findings have been reported in the literature review.

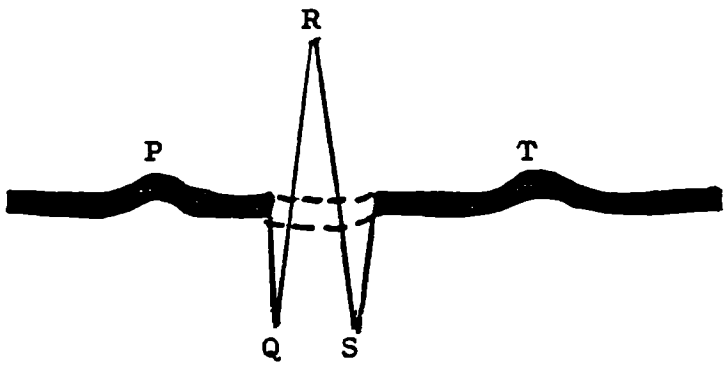
The electrodes used were silver/silver chloride and consist of a metal in contact with a solution of its own ions that minimizes polarisation potentials which contribute to drift and other sources of error. Electrodes were checked prior to use and only those electrode pairs having a resistance of less than 2 K were used. Since, skin conductance varies directly with effective electrode area and all measurements should be reported in terms of specific conductance i.e. in micromhos per square centimetre,

(Lykken & Venables, 1971), it is necessary to know the area of the skin surface being recorded. Although there is some controversy as to whether this relationship is linear (Venables & Christie, in Martin & Venables, 1980), the simplest way to control for this is to record from a constant known skin area. Therefore, electrodes of 1 cm diameter were used and carefully placed onto plastic shields with a 1 cm diameter hole so that a 1 cm area of the skin was recorded. Based on a survey of possible recording sites (Edelberg, 1967), the medial phalanges of the fore and middle fingers of the subject's non preferred hand were used. These sites were used as electrodes may easily be placed firmly onto them, they are relatively free of callouses and scar tissue, and are representatively electrodermally active. To allow good electrical contact an isotonic saline gel was employed (i.e. 0.05 Molar NaCl), as it is important that the electrolyte is compatible with the biological system in which it is in contact and human sweat varies from 0.015M to 0.06M (Rothman, 1954).

Heart Rate

Heart rate is relatively easy to measure as it consists of a series of discrete identifiable events. However, the biological significance of the heart as a pump which supplies oxygenated blood to the tissues of the body in accordance with their metabolic requirements is illustrated by the complexity of the underlying physiology and the homeostatic control of cardiac responding. This complexity presents problems to the psychophysiologist who wishes to use cardiac activity as a dependent variable, with respect to the selection of relevant measures, the appropriate

FIGURE 5. THE ELECTROCARDIOGRAM TRACE



P :- DEPOLARIZATION OF THE SINOATRIAL NODE
PRESSURE INCREASE IN ATRIA AND VENTRICLES
BLOOD FLOW FROM ATRIA TO VENTRICLES

PR :- EXCITATION OF ARTERIVENTRICULAR NODE
RELAXATION ATRIA
HIGHER PRESSURE IN VENTRICLES

QRS :- VENTRICULAR DEPOLARIZATION CAUSING CONTRACTION
RISE IN PRESSURE IN ATRIA AND VENTRICLES

T :- REPOLARIZATION AND RELAXATION OF VENTRICLES

method of analysis and also the interpretation of the results.

The mammalian heart consists of four chambers, two ventricles and two atria. The main function of the atria is to act as reservoirs for blood returning from the venous circulation prior to its reintroduction into the arterial circulation by pumping action of the ventricles. The right ventricle supplies blood to the lungs via the pulmonary circulation and the left ventricle supplies blood to the rest of the body tissues via the systematic circulation. The cardiac circulation is represented in Figure 5, and describes the sequence of events that occur during the expulsion of blood into the arterial circulation.

Heart rate describes only the time interval between consecutive ventricular contractions as indicated by the R wave of the electrocardiogram (ECG). This measure is the most commonly used, but provides only limited information about the cardiac cycle and its output. Other factors such as the amount of blood that enters the ventricles and the efficiency of the ventricular contraction also contribute to cardiovascular activity, and hence, need to be considered when interpreting heart rate changes and also in the evaluation of inconsistent cardiac data. The neural innervation of the heart is complex but can be separated into two interactive inputs deriving from the sympathetic and parasympathetic branches of the autonomic nervous system, (SNS and PNS respectively). Excitation of the SNS inputs results in increases in blood pressure and heart rate - the net result being faster ejection of blood from the ventricles and a slower filling of the atria. The

PNS input originates from the vagus nerve and produces changes that are essentially antagonistic to sympathetic activity and results in a decrease in heart rate. This is an oversimplification however as the degree of PNS inhibition is also a function of the level of SNS activity and there are also intrinsic control factors such as autoregulation due to the intrinsic properties of the cardiac muscle and the action of pressure sensitive baroceptors and also chemoceptors which are sensitive to the concentration of O_2 and CO_2 in the blood. Other gross influences include respiratory activity and body movement.

Against this background it is surprising that cardiac activity has been used so extensively as a psychophysiological variable. One of the reasons for this is due to the concept of 'arousal'. Cannon (1920), put forward the idea of a generalised physiological response which accompanied motivational changes elicited by a change in stimulation. Such a response was preparatory to the increased behavioural drive due to the stimulation (Duffy, 1962). The importance of the heart in meeting metabolic requirements of the organism led to the use of heart rate as an index of arousal. However, it is this biological importance with the associated homeostatic control that shows such a rationalisation as being oversimple. The expected increase in heart rate contingent upon stimulation is confounded by experimental findings of heart rate deceleration to simple stimuli (Darrow, 1929) and heart rate acceleration to intense or threatening stimuli (Sternbach, 1960). Both acceleration and deceleration responses have been observed in classical conditioning

paradigms (Wilson, 1969). The 'directional fractionation' hypothesis (Lacey, 1967) accounts for heart rate deceleration as being due to situations requiring environmental intake, and acceleration due to environmental rejections. Experimental studies have not provided unequivocal support (Siddle & Turpin, 1979). Obrist et al (1974), have proposed that changes in cardiac activity reflect changes in the level of somatic movement - the 'cardiac-somatic coupling' hypothesis. However, Obrist has also argued that under certain conditions e.g. those related to active avoidance of aversive stimuli, the cardiac-somatic coupling is dissociated, resulting in heart rate acceleration which is unrelated to the overt level of somatic activity.

In summary, there are three distinct approaches in using cardiac activity as an index of psychological state. Firstly, as an index of arousal, but there are problems due to lack of correlation between heart rate and other measures of arousal. Secondly, the Lacey hypothesis, which emphasises the role of cardiac activity as an intervening variable in information processing. Thirdly, Obrist's approach, which views phasic cardiac activity against the background of intrinsic control systems which regulate heart rate according to homeostatic requirements.

Sayers (1975), has suggested the use of long term measures of cardiac activity as phasic changes tend to be unreliable and when such changes do occur they are probably modified or attenuated by homeostatic constraints. This enables an estimate of variability in the resting state prior to stimulus intervention and also ensures that the heart rate response is actually measured. This is especially

important given the variability in both latency and direction of heart rate responses and maximises the available information in the recording.

Each heart beat is a well defined electro-mechanical event and is readily measured peripherally in several different ways. The first study used a finger photo-plethysmograph. This measures the amount of light transmitted through the finger or reflected by the finger, and the increased blood flow caused by the heart beat interferes with the light transmission or reflection affording an index of cardiac activity. Problems involved in such a device arise from sensitivity to bodily movement and also a variability in amplitude due to the respiratory cycle. The second study to be reported used electro-cardiography, a method that measures the electrical potentials associated with the cardiac cycle. The body acts as a volume conductor transmitting the electrocardiac signals to the body surface. Stainless steel plate electrodes were used, secured to the limbs with rubber straps and an hypertonic saline gel was used as a contact medium to maximise electrical contact. The placement sites were left arm and right leg for the active electrodes and the left leg for the ground electrode. The sites were reversed for left handed subjects. Such placement enables the control box to be manipulated without causing excessive movement artefact in the recording.

The raw datum is the interbeat interval (IBI), which is the time between two consecutive R spikes on the ECG. It is possible to express heart rate responding as beats per minute (BPM). That is IBI=60/HR, and also each measure

can be expressed either in terms of beat by beat, or second by second. There is no clear consensus which measure is the most appropriate, however, co-ordinating stimulus and cardiac events is simpler with real time units. A problem with this is the occurrence of differential responses due to where stimulus onset occurs in the cardiac cycle (Lacey & Lacey, 1974), but this effect is trivial if sufficient prestimulus measures are taken (Graham, 1979). Only when rate is estimated from real time units will the averaged data over the trial be unbiassed (Graham, 1979) so heart rate in beats per minute was derived second by second from the raw data.

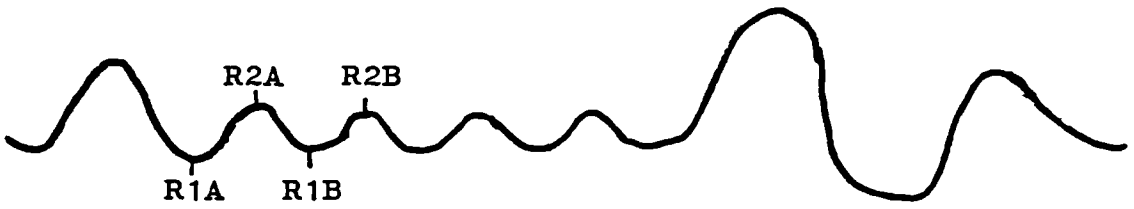
It is possible to derive many indices from the raw data (e.g. mean levels, variability scores), and as there are certain differences between the two experimental studies the relevant details are given in the appropriate chapter.

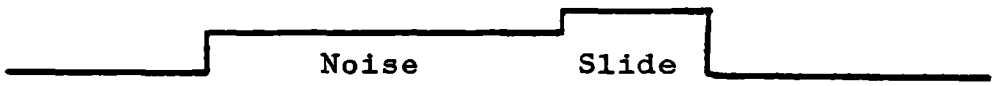
With the physiological considerations in mind and also the complexity of the experimental design in the learned helplessness paradigms (i.e. moving the manipulandum, the onset/offset of the aversive stimuli, the feedback slides) it is a debatable point whether heart rate is a useful measure in the learned helplessness research, even though the electrode placement is such as to minimise movement artifacts. However, both from the simple arousal hypothesis and from Obrist's (1976) cardiac-somatic hypothesis it is reasonable to predict that the differing degrees of aversiveness in the triadic design should be reflected in cardiac activity.

Respiration

Oxygen is essential for various processes to occur in the tissues and it is surprising that little work has been done using respiration as a psychophysiological variable either as a variable in its own right or because of the effect of respiration on other measures e.g. cardiac activity. Respiration rate varies with age - in the neonate human it is about 40 cycles per minute decreasing to about 15-20 cycles per minute in the adult human. Heart rate cycles of approximately five seconds duration are evident in most experimental subjects and the cause of this is sinum arrythmia. The mechanism of respiration is as follows - during inspiration the thoracic cavity is enlarged by the contraction of the diaphragm and the movement of the ribs and sternum upward effected by the intercostal muscles. The volume in the thoracic cavity increases, the pressure is reduced and the lungs expand so that air is drawn into them from outside. When the thorax goes back to its original position, pressure is put on the lungs and air is expelled. These movements are controlled by a respiratory centre in the medulla oblongata near the cardiac centre. The lungs are also innervated with branches of the vagus nerve, also important in cardiac control. This control system is essentially involuntary in that there are limitations on holding breath and breathing rate although voluntary control is possible within these confines. Breathing rate is adjusted to keep the percentage of CO_2 in the alveolar air at 5-6%.

FIGURE 6. THE RESPIRATORY TRACE





R1 TROUGH

R2 PEAK

60/(R1b - R1a) IN SECONDS = RESPIRATION RATE (cycles/min)

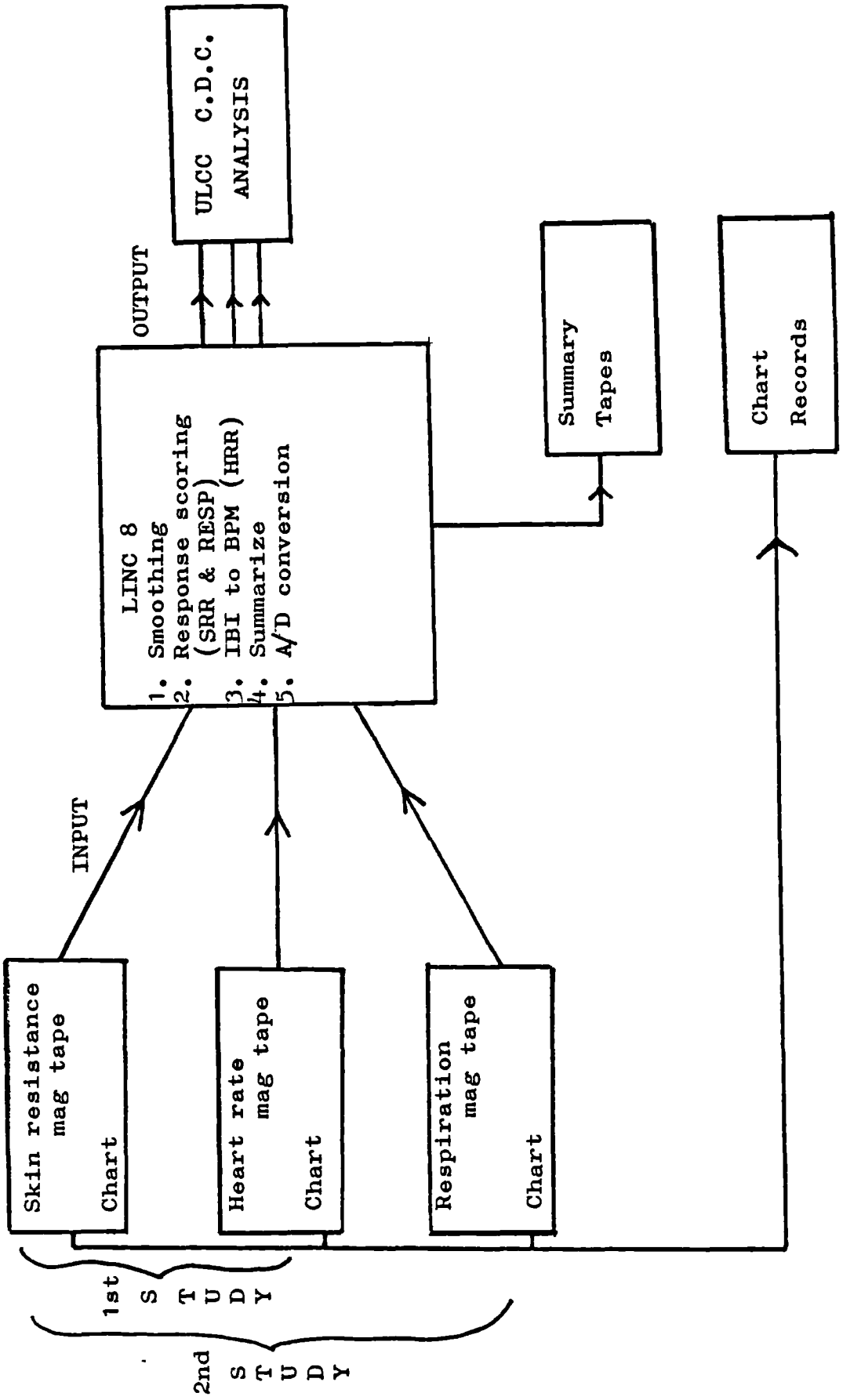
(R2a - R1a) / (R1b - R2b) = INSPIRATION/EXPIRATION RATIO

There are two main methods of measuring respiration. The first, uses a strain gauge placed around the subject's chest that records thoracic movement during respiration. The second method uses a thermistor placed under the subject's nose. The metal bead of the thermistor is heated when warm air is exhaled over it and cooled when inspiration takes place, due to the passage of cool air. This heating and cooling alters the electrical properties of the thermistor and can be recorded as a change in resistance. Measuring oxygen uptake by the subject requires costly equipment and so these two methods are favoured, but there are associated problems. Respiration rate does not directly reflect oxygen uptake - the obvious factor missing is the depth of respiration. The amplitude of the recorded respiration trace relates to respiration depth but without assuming linearity of resistance change in the thermistor contingent upon heating or cooling, the relationship is crude within subjects and of dubious validity across subjects. Similar arguments apply to the strain gauge.

Figure 6 shows an idealised respiratory trace in the second study in which respiration was measured using a nasal thermistor. Scoring peaks and troughs and relating these to time base affords measures of respiration rate, (cycles per minute) and inspiration/expiration ratio. These time base measures do not make assumptions about the electrical properties of the transducer.

The measure was taken to give information about the sinus arrythmia effects which may have affected the cardiac data collected in the first study which were very inconsistent; also, to investigate respiration as a

FIGURE 7. FLOWCHART OF DATA PROCESSING FOR THE PHYSIOLOGICAL VARIABLES



dependent variable in its own right. The nasal thermistor was selected as it is more sensitive than the strain gauge and it was thought that the phasic characteristics of the respiratory trace may be of some interest (Simpson, 1973).

Methodology and Analysis

The physiological variables used in the first study (SRR and HRR) were recorded on paper charts via an Elema Schonander Mingograph 800 and at the same time on magnetic tape via a Bell and Howell VR 3200 tape recorder (Figure 7). The variables from the second study (SRR, HR, and respiration) were recorded on paper charts via an Elema Schonander Mingograph 16 channel EEG recorder and on magnetic tape via a Hewlett Packard 3960 Instrumentation recorder. All data were fed into a LINC 8 computer to allow the isolation of the trial blocks (typically about 15-30 seconds each), smoothing to diminish artefacts due to mains interference, identification of response features, and the addition of stimulus markers where appropriate (e.g. conversion of response IBI to time base). These procedures were carried out using the SARA (Stimulus And Response Analysis) programme (Law, 1973). The data were summarized and printed out in digital format on paper tape to enable analysis on the ULCC CDC system. Details of the procedures differed between the two experimental studies and are reported in the appropriate chapters.

OVERVIEW OF PHYSIOLOGICAL VARIABLES

Against the background of unresolved issues, relating to what measures to take, problems of analysis, and the psychological significance of the measures given their

FIGURE 8. SUMMARY OF EXPECTED PHYSIOLOGICAL CHANGES FOR CONDITIONS OF STRESS AND NON-STRESS

	STRESSED	UNSTRESSED
SRR	1. Higher SCL across trials 2. More spontaneous responding 3. Larger amplitude responses 4. Attenuated Habituation	1. Lower SCL across trials 2. Less spontaneous responding 3. Smaller amplitude responses 4. 'Normal' habituation
HRR	1. Higher mean heart rate 2. More variability 3. Attenuated habituation	1. Lower mean heart rate 2. Less variability 3. 'Normal' habituation
RESP	1. Higher respiration rate 2. Presence of large sighs 3. Attenuated habituation	1. Lower respiration rate 2. Regular respiration 3. 'Normal' habituation

physiological complexity, a perspective is necessary to clarify the relevance of such measures to learned helplessness research and to derive some testable predictions.

The theory of learned helplessness predicts that uncontrollable aversive stimulation is more stressful than controllable stimulation and that repeated exposure to uncontrollable stimulation can lead to a depressive type state. It would be expected that this stress should be observed both in the self report data and in the physiological measures taken. The literature deals with findings from experiments designed to produce stress and anxiety in normals, and also a comparison between anxiety states and normals, schizophrenics etc. The former are the more relevant, but a clear distinction between state and trait anxiety is not always easy.

It has been found that anxiety neurotics showed a high skin conductance level and small responses (Solomon & Fentress, 1934) but Eysenck, S.G.B., (1956), was unable to differentiate between neurotics and normals. Lader & Wing, (1964, 1966), found less rapid SRR habituation to 100 db tones in anxious patients compared to normals, and they showed more non-contingent responding. Lader (1967), found slowest habituation in anxious/depressed compared to other patients, and Bond, James, and Lader (1974), found that anxious patients showed more non-contingent responding than normals during habituation trials. Using normal subjects, Miller & Shmavonian (1965), found skin conductance increases and a rise in non-contingent responding during anticipation of a painful stimulus. The present experimental studies involve a number of discrete trials where subjects attempt

to gain control over aversive noise. It is predicted that those subjects in the uncontrollable pretreatment should show increases in skin conductance level and more spontaneous electrodermal activity across trials compared to habituation trends (i.e. decrease in skin conductance level, less spontaneous electrodermal activity) in the other two groups.

An increase in heart rate has been reported to be associated with anxiety (Lader & Wing, 1966) and also increased cardiac variability (Whitehorn & Richter, 1937) and so it may be expected that during pretreatment a higher mean heart rate with greater variability will be shown in those groups exposed to uncontrollable contingencies.

Finesinger (1944), reported that anxious and depressed patients had increased sighing type respirations. Also, Goldstein (1964), found increased respiratory rate in anxious patients. Hence, higher respiratory rate which does not habituate may be predicted for the groups exposed to uncontrollable stimulation. This clinical evidence is of slight relevance and more relevant studies on the psychophysiology of controllability are given on pps. 46-48.

In summary, against the background of contradictory evidence, complex physiology, homeostasis, and individual differences, the subjects exposed to uncontrollable contingencies are expected to find the pretreatment more stressful. The physiological concomitants should be increased skin conductance levels with more spontaneous responding, higher respiratory rate and heart rate with more variability (Figure 8). There should also be an attenuation of habituation trends expected in the other two groups.

In the few helplessness studies which have used physiological measures the results are not clear and have already been reported in the literature review.

SUMMARY

Exposure to uncontrollability is more stressful than exposure to controllability and results in certain deficits in later learning. The stress induced by the pretreatment is assessed by physiological activity, mood check lists, and self report, whilst learning deficits can be identified on the anagram solving task. Other sources of variance can be attributed to individual differences and various measures relating to these are included.

Together with ratings of the stimuli etc. it should be possible to assess the effects of exposure to uncontrollable stimulation and to explore some of the cognitive components of the testing situation that contribute to the learned helplessness phenomenon.

CHAPTER 6

AN EXPLORATORY STUDY OF LEARNED HELPLESSNESS USING INSTRUCTIONAL SET AND NON-VERIDICAL FEEDBACK AS INDEPENDENT VARIABLES

INTRODUCTION

Introduction

The purpose of the first study was essentially exploratory, that is, to set up a learned helplessness experiment and to investigate possibilities for a larger study. This study was based on the triadic design of the Hiroto & Seligman (1975) studies, or rather on that experiment in the studies that used an instrumental pretreatment followed by a cognitive test task. In the Hiroto & Seligman experiment, college students were exposed to a pretreatment with inescapable, escapable, or passively tolerated aversive noise followed by an anagram solving test task. In the escapable pretreatment condition subjects could terminate the noise by pushing a button four times. In the inescapable pretreatment condition the buttom had no effect on the noise. Subjects were randomly assigned to one of three groups:-

a) ESCAPABLE - subjects who received 45 trials of unsignalled escapable noise.

b) INESCAPABLE - subjects who were yoked to the subjects in the escapable condition and received the identical trials of unsignalled noise.

c) CONTROL - subjects who passively listened to the identical number and duration of tones as the subjects in the other two groups.

The pretreatment consisted of 45 unsignalled trials with a 90 decibel tone @ 3,000 Hertz. If a subject in the escapable group failed to terminate the tone it lasted for 5 seconds and failure was signalled by the onset of a red

failure light. The intertrial interval (ITI) ranged from 10 to 25 seconds with a 14 second mean ITI. Triads of subjects in all groups received identical, yoked durations of the noise. After the pretreatments subjects rated the aversiveness of the tone. Subjects were then given 20 anagrams to solve, all of which were soluble and could be solved by using the same letter sequence.

The results from the Hiroto & Seligman experiment were in line with the theory of learned helplessness in that interference was produced by inescapability.

The study to be reported is similar to this experiment but with two major differences. Firstly, the subjects in the three experimental conditions were not yoked but exposed to identical series of tones, i.e. no group had control over the tones, but the escapable group were given feedback that they had controlled the tone (a green feedback light at the end of the trial). This seemingly trivial variant of the standard paradigm has been shown to be an effective manipulation (Geer, Davison & Gatchel, 1970) and is psychologically important (Miller, 1979), as it shifts the emphasis from objective control as defined by the experimenter to the non-veridical perception of control by the subject. It is also useful methodologically as all subjects in each condition are exposed to the same contingencies, whereas in the Hiroto & Seligman paradigm it is only triads of subjects across conditions that receive identical stimulus contingencies. Such a change enables comparison across subjects to be made more easily and more meaningfully.

The second change was that volunteers from the general public were recruited as experimental subjects as opposed to college students. This was due to the unavailability of college students at that time and an interest in whether the paradigm was sufficient given a more heterogeneous subject sample. In order to have some control over individual differences, the Eysenck Personality Questionnaire was administered together with the Mill Hill Verbal I.Q. Test (which is age corrected) to allow investigation of personality and I.Q. differences within such a subject sample.

The experiment to be reported involved an instrumental pretreatment where subjects in the escape condition were required to discover a pattern of button pushes and switches in order to terminate an aversive tone (95 decibel @ 600 Hertz). 'Success' feedback was given to subjects in the escape group at the end of the trial contingent upon four button pushes made during the noise. 'Failure' feedback was given to the subjects in the non-escape condition irrespective of any responding on the control box. Control subjects passively listened to the tones and were instructed to press the pushbutton four times at the onset of the noise, and that the end of trial would be signalled by a red light.

So, subjects in the escape and non-escape groups were set to initiate control over the noise and feedback indicated whether these subjects had in fact controlled the noise. Feedback indicating successful control over the noise was given only to escape subjects who had made four or more button pushes. Subjects in the passive control group were set to tolerate the noise and to press the button in order to "maintain attention".

It was predicted that subjects who were set to control the noise but were unable to do so i.e. the non escape group, would show deficits on the cognitive anagram solving task, and show patterns of physiological responsivity congruent with ratings of greater stress and anxiety. Referring back to the literature review, it can be seen that this study is a replication of the standard helplessness type experiment and it was intended that this replication would provide the basis to examine the "cure" and "immunization" predictions of the theory of learned helplessness. However, based on the results of this study and in conjunction with the reformulation of the original thoery the research took on to a different direction. These points will be discussed more fully in the chapter on the main study.

METHOD

Method

Subjects

Subjects were 30 volunteers from the general public, recruited by means of advertisements in the local newpapers. They consisted of 15 males and 15 females of mean age 35.3 years and 36.9 years respectively. Five males and five females were randomly assigned to one of the three experimental groups. Data relating to I.Q. and personality are given in Tables 13 and 14. All subjects received payment for their participation in the experiment.

Apparatus

The pretreatment phase of the experiment required subjects to attempt to control noise (95 decibel @ 600 Hertz), presented over a pair of headphones from a signal generator, by means of a control box. The control box measured 3" X 1" and consisted of a pushbutton and a toggle-switch. The subject held the control box in his dominant hand and was able to manipulate the button and switch using his thumb only, hence minimizing movement. A display panel of one green light and one red light was mounted on the wall in front of the subject to provide trial by trial feedback to the subject on his performance.

The test phase of the experiment consisted of a series of 20 soluble five letter anagrams selected by Hiroto & Seligman (1975) from the Tresselt & Mayzner (1966) list. The anagrams were presented on slides by means of a Kodak carousel slide projector, and were back projected onto a screen facing the subject. The slide projector was operated by a second experimenter who was seated in the room with the

subject, and who also timed the subjects anagram solving performance with a stopwatch.

Heart rate and skin resistance were recorded throughout the entire experiment. Silver/silver chloride electrodes, to record skin resistance, were attached to the ventral sides of the subject's first and second fingers of the non preferred. The use of chlorided electrodes minimizes shifts in resistance due to polarization, and electrodes were rechlorided if they were found to have a resistance of 2 KΩ or more across them. The use of plastic masks with 1 cm diameter holes (which correspond to the 1 cm diameter electrodes) ensured that the recording area was kept constant for every subject. An isotonic saline gel was used as a contact medium. A finger photoplethysmograph was attached to the third finger of the same nonpreferred hand in order to record heart rate. These physiological measures were recorded on paper chart via an Elemar Schonander Mingograph and also on magnetic tape via a Bell & Howell tape recorder.

Procedure

Subjects were randomly assigned to one of three experimental groups. The details of these groups are as follows:-

a) ESCAPE: Subjects in this group received 25 trials of unsignalled noise and were led to believe that they could reduce the duration of the noise by making a particular combination of button pushes and switches on the control box. Subjects were asked to discover the correct response (which was four button pushes), and told that this response would reduce the duration

of the noise to which they were exposed. Discovery of the correct response would be indicated by a green light on the display panel, whilst incorrect response patterns would be signified by a red light on the display panel. Responding had no effect on the noise duration, but did control the light display, so inducing the set of non-veridical perceived control.

b) NON-ESCAPE: Subjects in this group were given the same instructions as subjects in the escape group but any manipulation of the control box resulted in the failure signal (a red light) on the display panel. Subjects should develop the set of the perception of non-control over the situation.

c) PASSIVE-CONTROL: Subjects in this group were told to press the button four times when the noise came on, and that the red light indicated the end of the trial.

The instructions used are given in the appendix. Subjects in the first two groups were "set" to initiate controlling behaviour over the noise in the comtext of a delay in reinforcement experiment. Feedback imdicated that escape subjects had been able to control the noise and that non-escape subjects had failed to control the noise. Subjects in the third passive control group were not "set" in this way, but were simply asked to respond to the omset of the noise.

All subjects were seated in a comfortable chair and given a 5 second sample of the noise. Recording electrodes were then attached and the subject was asked to complete the following questionnaires:-

a) Eysenck Personality Questionnaire:- to identify atypical personality profiles, enable equivalence of personality scores across groups, and as an extra exploratory variable.

b) Mill Hill test of verbal I.Q.:- to allow I.Q. matched groups and a possible means of weighting the anagram test scores.

c) Nowlis Affect Check List:- to provide data on state mood changes across the various phases of the experiment.

Subjects were then given instructions appropriate to the experimental conditions to which they had assigned and left in the testing room for a five minute rest period. Twenty-five unsignalled tones were administered, which were identical in duration and inter trial interval for each group (mean duration = 10 seconds; mean inter trial interval = 25 seconds), achieved by means of on line use of a LINC 8 computer. The duration of the tones became progressively shorter to enhance the perception of control in the escape group. At the end of this phase the experimenter re-entered the room and asked the subject to complete another Nowlis Affect Check List. The subject was then told that the experimental test session was complete, but requested to do another experiment for another member of the department. All subjects agreed to stay on, and were introduced to the second experimenter, who sat next to the subject and after having given him/her a standard written set of instructions, presented the anagram test task. Time to solution was recorded by the second experimenter using a stopwatch, and noted onto a score sheet. If the subject failed to give a

correct answer within 100 seconds the next anagram was presented.

After the anagram test task the subject was asked to complete the final Nowlis Affect Check List. The first experimenter returned and administered a post experimental interview to the subject to obtain information about the subject's ratings of the noise, motivation to do well, and the effectiveness of the experimental treatments.

All subjects were fully debriefed and paid.

RESULTS

Anagram Results

Summaries of the anagram results are presented in Tables 1 and 2. These tables refer to results that have been broken down by group, experimental condition, and sex, and pooled across trials. Key 1 gives details of the group classification and also which groups constitute the experimental conditions and sex classifications. Key 2 describes the various measures taken from the raw data which are largely consistent with the measures taken in previous studies on learned helplessness. However, several points need to be discusseed. Firstly, subjects are allowed 100 seconds to solve each anagram and so, given that the subject has failed, the mean latency score can either include the failures as latencies of 100 seconds or exclude the failures and so reflect mean latency for solved anagrams only. This distinction has not been made clear in the previous literature and it seems that failures have been scored as 100 seconds each (as in the measure MLS 2). It is obvious that the measures are to some extent dependent and so it could be argued that MLS 1 (where failures are omitted) is more meaningful if the number of failures is also taken as a measure, as these two measures are independent. Both measures (MLS 1 & MLS 2) were computed in this study. Secondly, given that the helplessness deficits will be alleviated once the subject succeeds on the test task, then estimates of the deficits over 20 trials will be conservative. It would be expected that any helplessness deficits would be more significant over the initial trials of the test task. Hence, latency scores (both MLS 1 & MLS 2) and the number of failures were

computed for the first four trials only. Other measures (e.g. trials to criterion and the conditional probabilities) were not computed as they are not meaningful for the four trials only.

The majority of studies using the anagram solving test task have carried out non-parametric analyses e.g. Kruskall Wallis analysis of variance, and so this analysis was carried out on the data. All the results were statistically non-significant and given the greater statistical power of the parametric techniques, these analyses are not further reported. Consistent with Teasdale (1978), parametric analysis of variance was carried out on the data. Frequency distributions indicated that certain measures had skewed distributions (e.g. latency scores and the conditional probability scores) and so the appropriate data transformations were performed to "normalize" the data prior to analysis, latency scores were transformed to LOG seconds and the conditional probabilities were transformed using ARCSIN. Parametric analysis of variance was carried out on both the raw data and the transformed data in order to see whether the transformations had affected the variance estimates to a significant extent. As the transformations had little effect on the variance estimates and yet adjusted the frequency distribution of the scores, the results of the analysis on the transformed data are given at the foot of each variable table. The results show a statistically significant sex difference on some of the anagram variables (i.e. MLS 2, FAILURES), there is a statistically significant group effect, experimental condition effect, and sex by experimental condition

interaction. Referring back to the tables of group means, it seems that these effects are due to the superior performance of the group G2 (males in the escape condition). Referring to the means for each experimental condition it can be seen that for most measures the non-escape groups did worse than the escape groups but these results did not reach statistical significance in general. Also, it can be seen that the passive control groups did worst on most measures. These results will be discussed in some detail and it is worth pointing out the large standard deviations associated with these measures and the large error variance estimates against which these effects were tested.

Finally, all measures and the verbal I.Q. scores of subjects were intercorrelated (Table 3), and it can be seen that the anagram measures correlate positively and significantly with each other, and that these measures correlate negatively and significantly with the verbal I.Q. scores. Despite this significant I.Q./anagram measure correlation, the subjects were fairly homogeneous on the I.Q. scores per se. Against the background of small group effects and large error variance it was found that adjusting the anagram scores on the basis of verbal I.Q. made very little difference. Also, the anagram scores were obtained after exposure to the pretreatment phase of the experiment. Adjustment would be justified on the basis of data from the I.Q. test together with anagram test scores obtained prior to the pretreatment but these data were not available.

KEY 1. KEY TO GROUP LABELLING FOR TABLES

GROUPS (n=5)

G1 NON-ESCAPE MALES

G2 ESCAPE MALES

G3 PASSIVE CONTROL MALES

G4 NON-ESCAPE FEMALES

G5 ESCAPE FEMALES

G6 PASSIVE CONTROL FEMALES

EXPERIMENTAL CONDITIONS (n=10)

H1	NON-ESCAPE	(G1,G4)
H2	ESCAPE	(G2,G5)
H3	PASSIVE CONTROL	(G3,G6)

SEX (n=15)

P1	MALES	(G1,G2,G3)
P2	FEMALES	(G4,G5,G6)

KEY 2. KEY TO DEPENDENT VARIABLES FROM THE ANAGRAM SOLVING TASK

MLS1 :- MEAN LATENCY SCORE FOR SOLVED ANAGRAMS IN SECONDS.

MLS2 :- MEAN LATENCY FOR ALL ANAGRAMS (FAILURES TO SOLVE COUNTED AS 100 SECONDS) IN SECONDS.

FAILURES:- MEAN NUMBER OF FAILURES TO SOLVE WITHIN 100 SECONDS.

TC :- NUMBER OF TRIALS TO CRITERION OF THREE CONSECUTIVE SOLUTIONS IN 5 SECONDS EACH.

CP :- CONDITIONAL PROBABILITY OF SOLUTION GIVEN THAT THE PREVIOUS ANAGRAM WAS SOLVED.

TABLE 1. THE RESULTS FROM THE ANAGRAM SOLVING TASK

(20 ANAGRAMS)

	MLS1		MLS2		FAILURES	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
G1	15.00	6.47	27.82	19.40	3.2	3.42
G2	6.72	3.44	9.44	8.11	0.6	1.34
G3	20.31	11.51	41.22	13.01	5.4	1.52
G4	19.43	12.12	32.57	25.27	3.2	3.42
G5	20.38	11.98	40.68	20.24	5.3	3.36
G6	17.42	10.71	38.16	25.61	5.4	4.56
H1	17.22	9.16	30.20	21.24	3.2	3.22
H2	13.55	8.31	25.06	14.54	3.0	2.41
H3	18.87	10.48	39.69	19.15	5.4	3.20
P1	14.01	7.29	26.16	14.22	3.1	2.12
P2	19.08	10.76	37.14	22.07	4.7	3.54

	SOURCE	F	d.f.	p
MLS 1	G	1.71	5.24	N.S.
	P	1.71	1.24	N.S.
	H	1.33	2.24	N.S.
	H X P	2.11	2.24	N.S.
MLS 2	G	3.76	5.24	$<.025$
	P	3.79	1.24	$<.10$
	H	2.79	2.24	$<.10$
	H X P	4.42	2.24	$<.025$
FAILURES	G	3.51	5.24	$<.025$
	P	3.80	1.24	$<.10$
	H	2.92	2.24	$<.10$
	H X P	3.96	2.24	$<.025$

TABLE 1. (contd)

	TC		CP	
	MEAN	S.D.	MEAN	S.D.
G1	12.4	7.7	0.898	0.10
G2	6.2	3.1	0.976	0.05
G3	11.2	6.2	0.702	0.14
G4	13.6	5.4	0.874	0.25
G5	14.8	5.8	0.790	0.18
G6	11.6	7.9	0.728	0.34
H1	13.0	6.3	0.886	0.18
H2	10.5	4.4	0.883	0.13
H3	11.4	6.7	0.715	0.24
P1	9.93	5.5	0.859	0.10
P2	13.33	5.9	0.797	0.25

	SOURCE	F	d.f.	p
TC	G	1.14	5.24	N.S.
	P	2.23	1.24	N.S.
	H	0.41	2.24	N.S.
	H X P	1.32	2.24	N.S.
CP	G	0.86	5.24	N.S.
	P	1.15	1.24	N.S.
	H	1.27	2.24	N.S.
	H X P	0.31	2.24	N.S.

TABLE 2. THE RESULTS FROM THE ANAGRAM SOLVING TASK (FIRST 4 ANAGRAMS ONLY)

	MLS1		MLS2		FAILURES	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
G1	24.07	31.97	43.05	43.49	1.0	1.0
G2	16.77	21.30	25.10	32.59	0.4	0.8
G3	21.25	24.30	52.75	43.68	1.6	0.6
G4	40.54	32.70	61.35	39.01	1.4	0.8
G5	24.73	20.90	58.60	41.30	1.8	1.1
G6	22.55	29.04	65.15	43.79	2.2	1.6
H1	31.72	31.71	52.20	40.78	1.2	0.9
H2	19.79	20.77	41.85	36.72	1.1	0.9
H3	21.81	25.73	58.95	43.17	1.9	1.2
P1	20.39	25.46	40.30	39.57	1.0	0.8
P2	30.37	27.36	61.70	40.71	1.8	1.2

	SOURCE	F	DF	P
	G	2.96	5.24	<.05
MLS1	P	3.21	1.24	<.10
	H	1.49	2.24	N.S.
	H X P	4.31	5.24	<.01
	G	1.19	5.24	N.S.
MLS2	P	.01	1.24	N.S.
	H	1.39	2.24	N.S.
	H X P	1.58	5.24	N.S.

TABLE 3. CORRELATION MATRIX OF ANAGRAM SCORES AND I.Q. TEST SCORES

	I.Q.	LATENCY	FAILURES	TC
I.Q.	1.00	-0.38 *	-0.48 **	-0.38 *
LATENCY		1.00	+0.83***	+0.79***
FAILURES			1.00	+0.79***
TC				1.00

* $p<.05$

** $p<.01$

*** $p<.001$

Skin Conductance Results

The skin conductance data were recorded on magnetic tape and, by use of the SARA program (Law, .1973), 25 blocks of data of 12 seconds length each were isolated by the LINC 8 computer system. An idealised data block is shown in Key 3, which illustrates the measures taken relative to the time based stimulus presentation. The main variables of interest are the basal levels which are tonic levels of autonomic activity, and the number of responses that occur against this tonic resting level. These may be regarded as measures of the subject's arousal and are most commonly used in the literature. Measures of response amplitude and rise time were also taken and are of secondary importance.

The raw data on the magnetic tape were converted to digital format via the LINC 8 and represented in the CDC computer system in resistance (kilohms). For statistical reasons and more importantly for biological reasons previously outlined, the data were transformed into conductance by dividing each resistance score into 1000 (i.e. micromhos). Two subjects contributed extremely low scores compared to the other subjects and this was most likely to be due to a methodological error, (for example, an electrical short between the recording sites of the adjacent fingers). The data were analysed for all 30 subjects and also for 28 subjects. Exclusion of these two outliers made no difference to the F ratios or to the statistical significance of the results, but as the reasons for these scores are more likely to be due to experimental error rather than naturally occurring extreme scores, the results are based on 28 subjects.

Frequency distributions of the basal levels and response amplitude measures showed that the distributions were skewed, and scattergram plots showed that the standard deviation of the scores was proportional to the square of the mean, and so a log transformation was carried out (Maxwell, 1958), prior to analysis. The data were analysed using an analysis of variance program VAR3 (Lebeaux, Lepine, Rouanet, 1975) and the key to the analysis is given Key 4. The results from the analysis are given in Tables 4 to 9 and a summary of these results is as follows:-

a) Onset basal level (Table 4): Collapsing all groups there is no significant trials effect, but there is a significant difference between groups across trials (i.e. G.T $p<.025$). This can be broken down into a sex difference (P $p<.1$) especially evident across trials (P.T $p<.025$) and an effect due to experimental condition across trials (H.T $p<.001$) and not due to experimental conditions per se (H p N.S.). When the usual linear trends are taken no differences emerge; however, when the less conservative estimates are taken there is a significant linear tremd ($p<.001$) and differences between groups on linear trends ($p<.001$).

Males maintained a higher skin conductamce level over all the trials compared to the females, (Graph 1), and the basal level changes do not differ appreciably across trials between sexes (Graph 1). The plots of basal level by experimental condition (Graph 2) reveal that the escapable group and passive control groups show a trend toward reduced skin conductance level

across trials, whilst the non escape group shows a trends towards increased skin conductance level across trials. Also, the escape group shows the highest skin conductance level initially, which the non escape group exceeds by trial 10. This result suggests that the non escape group became more aroused as the experiment proceeded whilst the other two groups showed a decrease in arousal indicative of habituation.

b) Offset basal level (Table 5): This measure is similar to the onset basal level measure but the group effects are attenuated due to the activity that occur during the trial. There is still a sex effect, but G.T, P.T & H.T effects are obscured by the greater variance in the scores generally, which is reflected in the significant trials effect ($p<.005$). The results are mainly the same for the offset basal level change as they were for the onset basal change.

c) Number of responses (Table 6): This was computed as the total number of skin conductance responses per trial block. Collapsing trials, the groups are significantly different (G $p<.01$), and collapsing groups the trials are significantly different (T $p<.001$). There is a significant sex difference (P $p<.05$) with males showing more responses than females, and a significant experimental condition effect (H $p<.05$) with the non escape group showing most responses and the passive control group showing the least. There is also a significant linear trend (LIN T $p<.005$) reflecting a general decrease in the number of responses across trials which is not

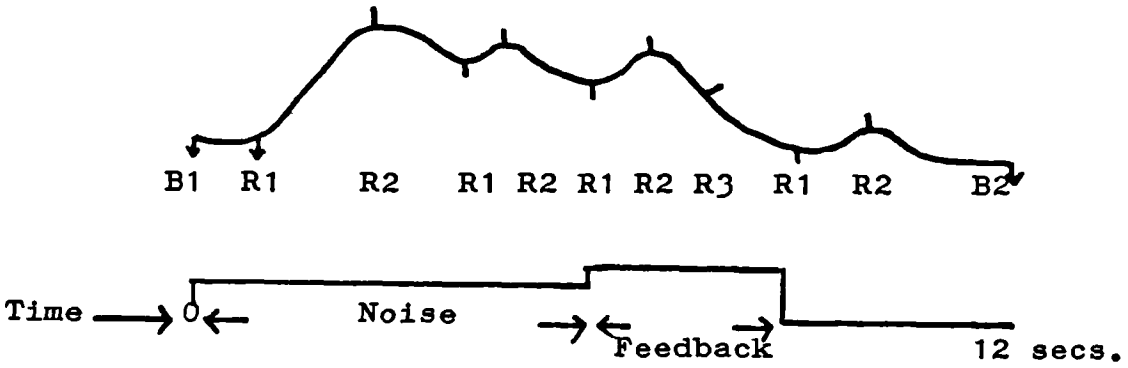
differentiated by group, or sex, and only to a small degree by experimental condition (H.T $p<.1$).

d) Response amplitude (Table 8): Response amplitude is the amplitude of the response that occurs upon trial onset (defined as 1.5 to 3.5 seconds after trial onset). There is a significant trials effect (T $p<.001$) with groups collapsed and this is probably due to habituation. Given the large error variance these habituation trends are not reflected in other components of the analysis, that is, the trials differ significantly but not in a consistent pattern.

e) Rise time (Table 9): This measure shows a similar pattern to the response amplitude measure, i.e. a significant difference between trials (T $p<.001$) with no clear consistency save for a significant linear trend (LIN T $p<.025$) indicative of habituation.

These last two measures were taken as it was predicted that there would be differential habituation trends across experimental conditions, which was not found in these data. The more meaningful measure of recovery of the response was precluded as the vast majority of responses either did not recover, or were sufficiently confounded by further responding to disallow accurate scoring of the recovery limb.

KEY 3. KEY TO SKIN CONDUCTANCE MEASURES



ONSET BASAL LEVEL : (B1) tonic skin conductance level taken immediately prior to trial onset (micromhos).

OFFSET BASAL LEVEL: (B2) tonic skin conductance level taken at the end of the trial (micromhos).

RESPONSE AMPLITUDE: (R2-R1) amplitude of phasic skin conductance response to onset of trial (micromhos).

RISE TIME : (R2-R1 time taken for response to reach peak amplitude (seconds).

N. OF RESPONSES : number of responses that occur within each trial block.

KEY 4. KEY TO SKIN CONDUCTANCE ANALYSIS

G : differences between groups (T collapsed).

T : differences between trials (G collapsed).

G.T
T.GW : differences between groups across trials.

P : differences between sexes (H & T collapsed).

P.T : differences between sexes across trials.

H : differences between experimental conditions (P & T collapsed).

H.T : differences between conditions across trials.

H.P : sex/condition interaction (T collapsed).

H.P.T : sex/condition/trial interaction.

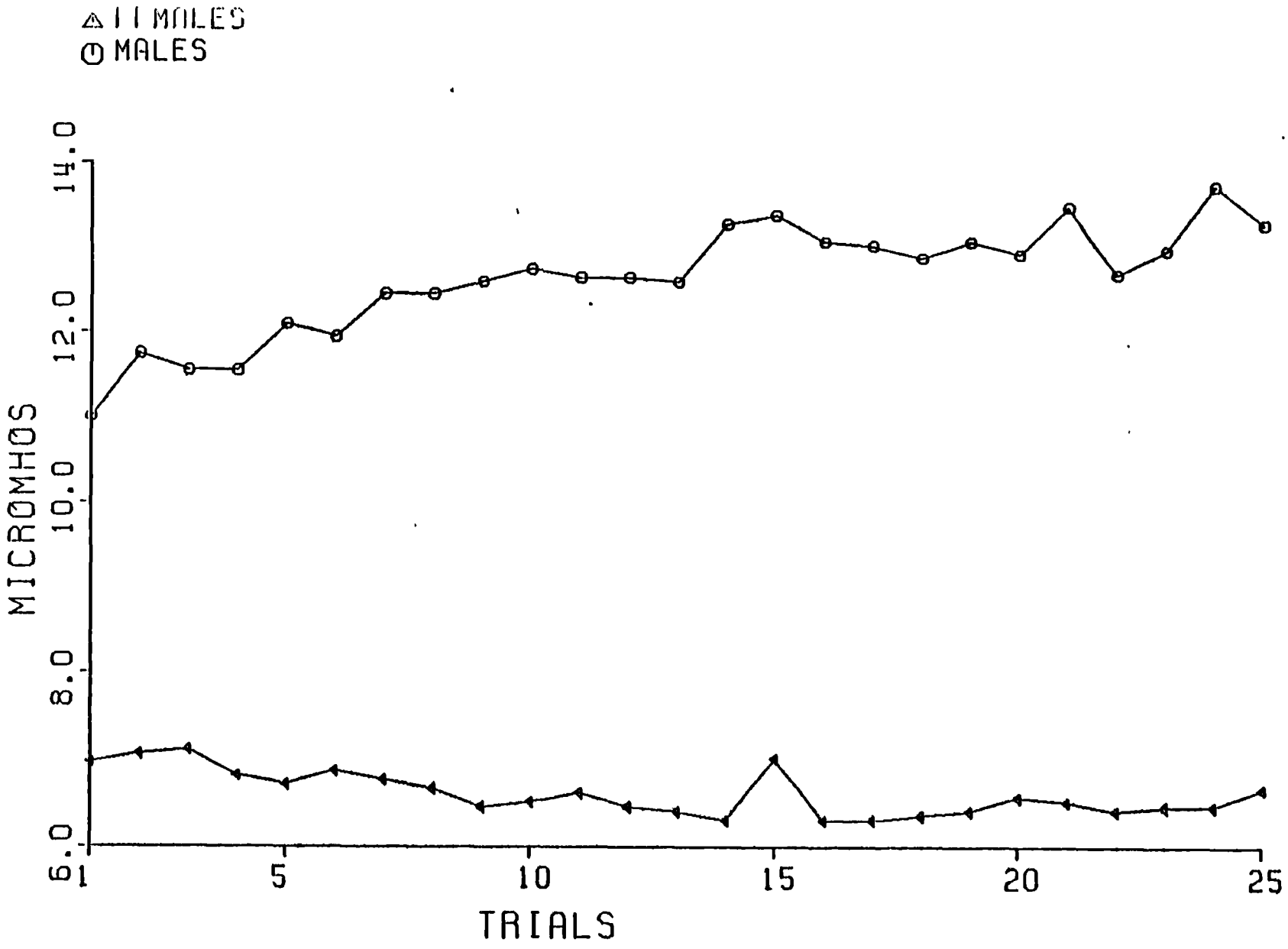
LIN T : trend slope different from zero (G collapsed).

G.W : differences between groups on LIN T.

TABLE 4. SKIN CONDUCTANCE:- ONSET BASAL LEVEL ANALYSIS (LOG MICROMHOS)

SOURCE	F	df	p
G	0.73	5,22	N.S.
T	1.25	24,528	N.S.
G.T	1.28	120,528	<.025
P	3.12	1,22	<.10
P.T	1.75	24,528	<.025
H	0.14	2,22	N.S.
H.T	1.97	48,528	<.001
H.P	0.18	2,22	N.S.
H.P.T	0.52	48,528	N.S.
LIN T	1.1 14.7	1,22 1,528	N.S. <.001
LIN T G.W	1.46 19.59	5,22 5,528	N.S. <.001

GRAPH 1: SKIN CONDUCTANCE ONSET BASAL LEVEL BY SEX



GRAPH 2: SKIN CONDUCTANCE ONSET BASAL LEVEL BY CONDITION

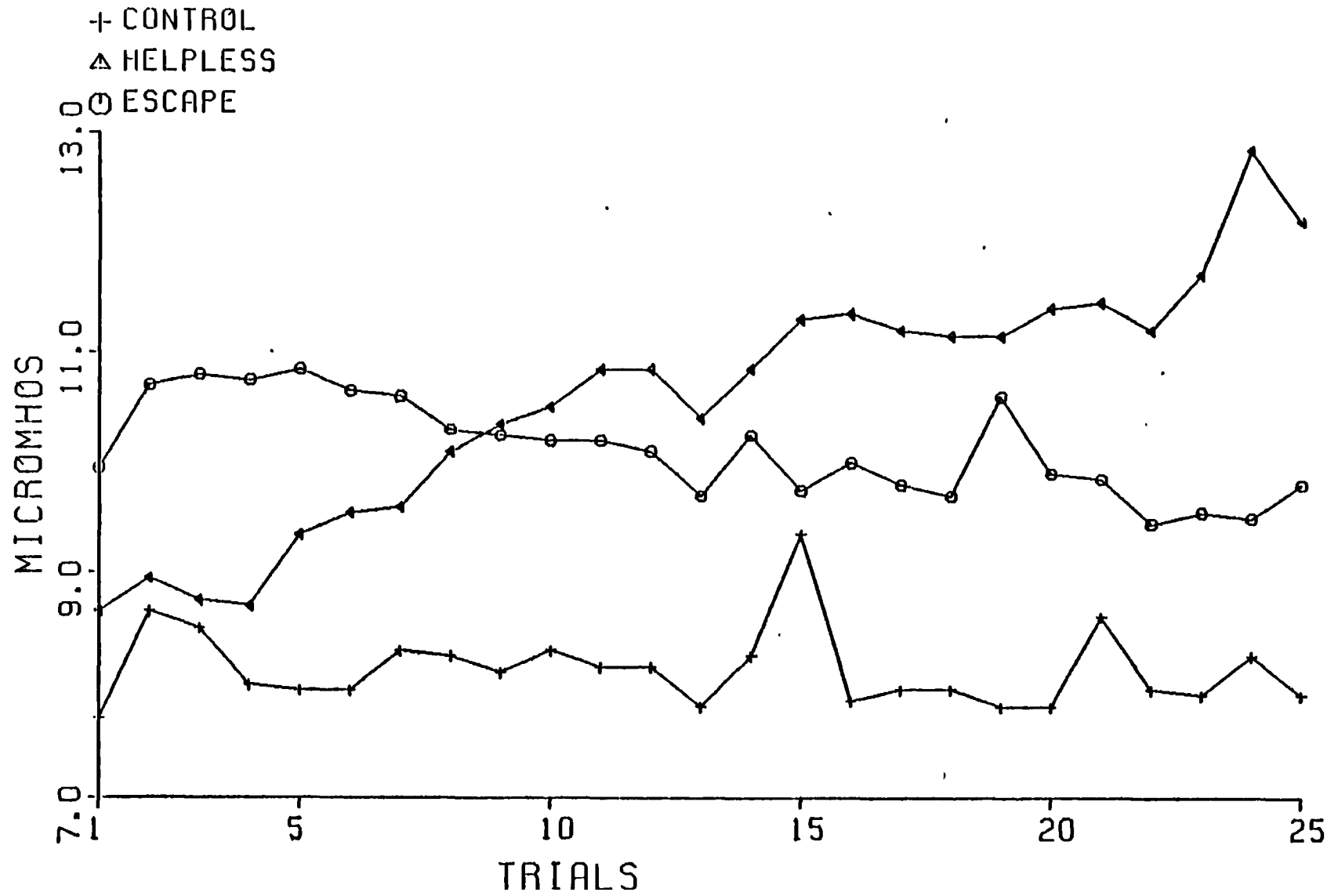


TABLE 5. SKIN CONDUCTANCE:- OFFSET BASAL LEVEL ANALYSIS (LOG MICROMHOS)

SOURCE	F	df	p
G	0.72	5,22	N.S.
T	1.97	24,528	<.005
G.T	1.00	120,528	N.S.
P	3.01	1,22	<.1
P.T	1.26	24,528	N.S.
H	0.19	2,22	N.S.
H.T	1.21	48,528	N.S.
H.P	0.18	2,22	N.S.
H.P.T	0.71	48,528	N.S.
LIN T	2.62	1,22	N.S.
	29.32	1,528	<.001
LIN T G.W	1.00	5,22	N.S.
	11.19	5,528	<.001

TABLE 6. SKIN CONDUCTANCE:- NUMBER OF RESPONSES

		TOTAL	MEAN
G1	=	262.0	2.096
G2	=	194.0	1.552
G3	=	186.0	1.860
G4	=	158.0	1.580
G5	=	171.0	1.368
G6	=	111.0	0.888
P1 males	=	642	1.836
P2 females	=	440	1.279
H1 helpless	=	420	1.830
H2 escape	=	365	1.460
H3 control	=	297	1.374

TABLE 7. SKIN CONDUCTANCE:- NUMBER OF RESPONSES ANALYSIS

SOURCE	F	df	p
G	4.48	5,22	<.01
T	3.23	24,528	<.001
G.T	0.96	120,528	N.S.
P	12.33	1,22	<.005
P.T	0.71	24,528	N.S.
H	3.86	2,22	<.05
H.T	1.28	48,528	<.1
H.P	1.94	2,22	N.S.
H.P.T	0.77	48,528	N.S.
LIN T	13.9 53.74	1,22 1,528	<.005 <.001
LIN T G.W	0.19 0.74	5,22 5,528	N.S. N.S.

TABLE 8. SKIN CONDUCTANCE:- RESPONSE AMPLITUDE ANALYSIS. (LOG MICROMHOS)

SOURCE	F	df	p
G	0.45	5,22	N.S.
T	2.67	24,528	<.001
G.T	0.92	120,528	N.S.
P	1.18	1,22	N.S.
P.T	0.77	24,528	N.S.
H	0.13	2,22	N.S.
H.T.	0.79	48,528	N.S.
H.P	0.34	2,22	N.S.
H.P.T	1.06	48,528	N.S.
LIN T	2.43 5.71	1,22 1,528	N.S. <.001
LIN T G.W	0.72 1.68	5,22 5,528	N.S. N.S.

TABLE 9: SKIN CONDUCTANCE:- RISE TIME ANALYSIS.
(SECONDS)

SOURCE	F	df	p
G	0.83	5,22	N.S.
T	2.83	24,528	<.001
G.T	1.09	120,528	N.S.
P	0.07	1,22	N.S.
P.T	1.09	24,528	N.S.
H	1.60	2,22	N.S.
H.T	1.01	48,528	N.S.
H.P	0.40	2,22	N.S.
H.P.T	1.17	48,528	N.S.
LIN T	6.47	1,22	<.025
	15.86	1,528	<.001
LIN T G.W	0.27	5,22	N.S.
	0.66	5,528	N.S.

Heart Rate Results

Heart rate was measured by using a finger photoplethysmograph placed on the third finger of the non-preferred hand and recorded on paper chart as well as on magnetic tape. Twenty second trial blocks were isolated for each trial by means of the Sara Programme (Law, 1973), on the LINC 8 computer system. The fundamental datum is the inter-beat interval, which is the time between each successive pair of R wave spikes in the trace, and is represented in histogram format beat by beat on the LINC display. To enable event related responses to be examined across subjects, the inter-beat interval was transformed to heart rate in beats per minute, second by second by referring the inter-beat intervals to a time base on the stimulus marker channel. The resulting data were mean level of heart rate in beats per minute for the 10 seconds prestimulus and mean heart rate level, second by second for 10 seconds post-stimulus. This is illustrated in Figure 9.

Two levels of responding can be identified:-

a) Phasic Responses to discrete stimuli e.g. the onset of the noise signifying the beginning of the trial. Such responses tend to be diphasic (i.e. an acceleration phase followed by a period of marked deceleration Lacey, 1967), which is presumably a function of the homeostatic control of the cardiovascular system.

b) Tonic Level Changes e.g. a change in heart rate level that is maintained over trials.

It might be predicted that differential habituation trends of the phasic responses and changes in tonic level would discriminate between experimental conditions, that is, the groups exposed to uncontrollable aversive stimulation would show an attenuation of habituation of phasic responses and a trend towards increased heart rate level over trials indicative of greater arousal/anxiety. In accordance with this and the complex homeostatic control mechanisms, a variety of measures were derived from the raw heart data to enable an exploration of this variable (Key 5). The measures were selected from many cited in Rolfe, (1973), and are:-

1. V1 Prestimulus Mean Level: this is the mean level in beats per minute for the 10 seconds prior to stimulus onset for each trial. Tonic changes would be reflected in this measure which also affords a range correction facility for poststimulus data points.

2. V2 to V10 Poststimulus Mean Levels: these are poststimulus mean levels in beats per minute, second by second, for 10 seconds after the stimulus onset. Phasic responding should be reflected in these points.

3. V12 to V21 Poststimulus Mean Levels (Law of initial values corrected): these are the same measures as V2 to V10 but each component is expressed as a function of the appropriate prestimulus mean level (V1), affording a law of initial values correction which takes some consideration of individual differences and tonic level changes that may affect phasic responsivity.

4. V22 Poststimulus Mean Level: this is the mean level in beats per minute for the whole 10 seconds post stimulus,

and again should reflect tonic changes as well as phasic activity.

5. V23 Standard Deviation of V2 to V11: this is an index of the variability of the 10 second by second scores that make up the poststimulus mean level (V22). Unless the response latencies are equivalent, the phasic responses may be eliminated when trials are pooled across subjects. So, this measure gives an index of heart rate change within each trial.

6. V24 Minimum Poststimulus Mean Level: this is the maximum deceleration observed in each trial in beats per minute and is an index of phasic responding which may be masked if data are pooled across subjects second by second.

7. V25 Maximum Poststimulus Mean Level: this is the same measure as V24 but is the maximum acceleration observed in each trial.

8. V26 (V25-V24): this is the difference between the maximum acceleration second minus the maximum deceleration second, and affords an index of maximum heart rate change in the poststimulus seconds of the trial in beats per minute.

9. V27 (V25 - Maximum Deceleration Second Following): this measure differs from V26 in that it measures the maximum heart rate change within a diphasic response and not the maximum change that occurs within the trial. For many trials, however, this measure will be the same as V26.

10. V28 (V1 - V22): this is the change in heart rate in beats per minute from the prestimulus mean level over 10

seconds to the poststimulus mean level over 10 seconds, and will reflect changes in tonic level.

These measures are illustrated in Key 5. The data were analysed by means of the VAR 3 analysis of variance programme (Lebeaux, Lepine & Rouanet, 1975).

There is a fairly consistent response to the onset of the noise but the exact nature of this response is variable. That is, it appears from the LINC display that for some trials an initial accelerative phase is followed by cardiac deceleration, but, for other trials this pattern of responding is reversed. This could be explained in terms of the fractional differentiation hypothesis (Lacey & Lacey, 1974), but one would expect that differences in stimulus intake or rejection would be reflected more consistently by subjects within a group, and not so much by trials within a given subject. Another hypothesis is that the nature of the heart rate response may be due to the position in the respiratory cycle where stimulus onset occurs. That is, the stimulus contingent heart rate response is confounded with the sinus arrythmia. As no measure of respiration was taken in this study, this hypothesis cannot be investigated. However, there are consistent heart rate responses but the characteristics of these responses are variable which would lead to these responses cancelling each other out when the data are averaged across subjects. It is likely that these effects, together with biological limitations on this response system could explain the largely non-significant results from the analysis.

Heart Rate Analysis

Summary tables of the analyses are not given, as the results are mainly non-significant. A discussion of the analyses is as follows. The analyses are best discussed by clustering the heart rate measures, that is:-

1. Tonic Mean Levels: V1, V22, V28. These measures reflect tonic levels and tonic level changes in heart rate responding.
2. Phasic Mean Levels: V2 to V11, V12 to V21. These represent indices of phasic heart rate responding.
3. Change Measures: V23, V24, V25, V26, V27. These represent change scores and variability scores of heart rate responding.

1. Tonic Mean Levels: The prestimulus mean level (V1) shows a highly significant trials effect (T) and a significant linear trend across trials (LIN T), but there are no significant differences evident when these effects are broken by group (G), sex (P), or condition (H). Similarly, all main effects and interactions are non-significant. The poststimulus mean level for 10 seconds (V22), shows a similar pattern, but the group X trial interaction (G.T), and sex X trial interaction (P.T) effects approach significance ($p<.1$), and the condition X trial interaction reaches significance ($p<.025$). This appears to be due to the male passive control group which maintains a higher level across all trials. This effect does not reach significance for the prestimulus mean level (V1), presumably because a small change in the F ratio has a fairly profound

effect on the statistical significance given the degrees of freedom involved (48,432). There is a highly significant condition effect (H), for V28, (the prestimulus mean level - the poststimulus mean level), which is also due to the passive control group males, who show greater change in mean levels across trials than the other two groups. These changes do not form a consistent trend (LIN T N.S.).

2. Phasic Mean Levels: The relevant variables are the poststimulus second by second mean levels (V2 to V11), and these scores expressed as a function of the relevant prestimulus mean levels (V12 to V21).

For V2 to V11, there are no significant main effects for condition, sex, or group but there are significant group by trial, condition by trial, and sex by trial effects. These are again reflections of the passive control group males who show a flatter trend with greater departures from the mean than the other two groups.

Similarly for V12 to V21, there are significant results for sex and experimental condition whilst the effects for trials have been reduced, presumably attenuated by the range correction of expressing scores as a function of prestimulus level. It would seem that the passive control group males are responsible for the statistically significant results found in the analyses.

3. Change Scores: V23 to V28. These measures relate to difference measures of heart rate change that occur within the 10 seconds poststimulus and again show a pattern of significant results against a background of non-significant results which appear to be due to the greater variability

of the passive control group males.

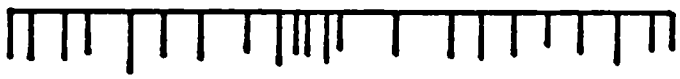
In summary, it appears that heart rate activity is not of great interest in this study. V22 (poststimulus mean level) indicates that the groups are relatively homogeneous and show a slight trend towards heart rate acceleration over trials. The passive control group males are exceptional as they maintain a higher level throughout the entire experiment, with no consistent trend save for that of greater variability. This is not a function of an outlier, but is a group characteristic for which there is no ready explanation. It is unlikely that these results are due to experimental factors unless they were effective prior to the pretreatment e.g. due to the instructions. There are no clues in the miscellaneous data that indicate that this group differs from the other groups on any dimension that could affect heart rate. It seems most likely that these results are due to the random allocation of subjects to experimental conditions, i.e. that a group has been selected that is atypical in basal heart rate level and cardiac responsivity by chance. Apart from this, the results are largely non-significant and this is probably due to several factors. Firstly, the tight homeostatic control over cardiac activity imposes tight constraints on responsivity. Secondly, sinus arrythmia effects may effectively produce differential response patterns that cancel out responses when averaged across trials or across subjects. Thirdly, either the situation was too complex to enable the identification of discrete cardiac responses, or, the situation was insufficient to produce cardiac responses at all.

It seems that the experimental conditions were sufficient to produce responses which were identifiable when the raw data were being processed, also, in producing significant SRR data etc. It remains to be seen whether these responses will discriminate between groups if information is available concerning sinus arrythmia, and the inclusion of a measure of respiratory activity in the second study should enable this to be investigated.

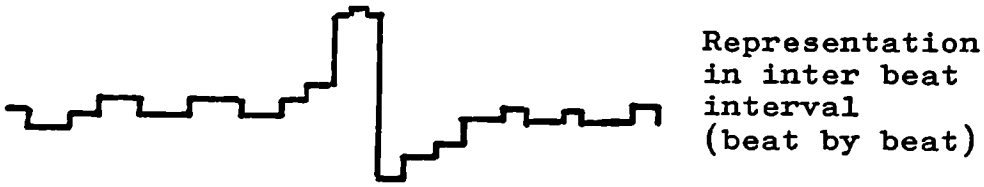
In conclusion, the heart rate data are of limited interest in this study, and unless the added variables in the second study prove otherwise, may show that the heart rate response variable is not a meaningful measure for studies that involve the complex testing situation implicit in the learned helplessness paradigm.

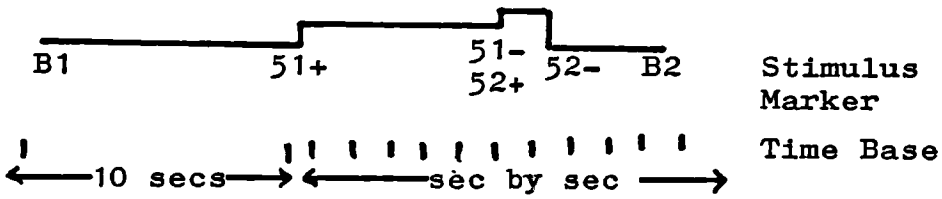
FIGURE 9. SCHEMA FOR HEART RATE DATA PROCESSING

A) CHART RECORD

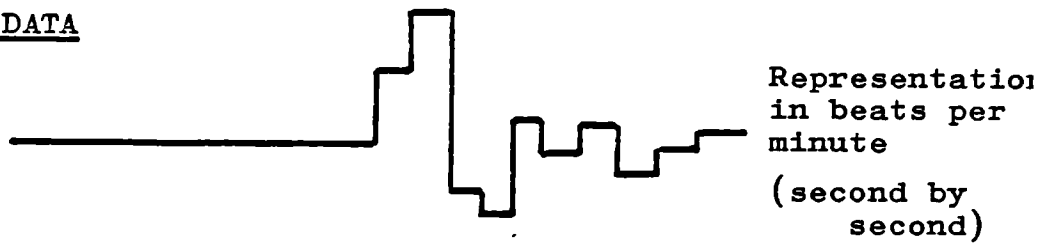


B) LINC DISPLAY

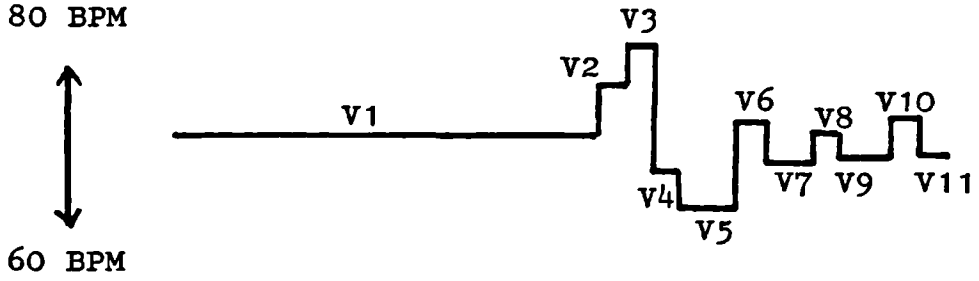




C) CONVERTED
LINC DATA



KEY 5. KEY TO HEART RATE MEASURES



V1 to V11	are mean level measures as shown.
V12 to V21	are $V2/V1$, $V3/V1$ ..... $V11/V1$ respectively.
V22	is (V2 + V3 + V4 ..... V11) / 10.
V23	is the standard deviation of V2 to V11.
V24	is the lowest BPM score (V5 in this case).
V25	is the highest BPM score (V3 in this case).
V26	is the highest minus the lowest (V3 - V5) for the entire trial.
V27	is the highest minus the lowest (V3 - V5) for the diphasic response.
V28	is the prestimulus mean minus the poststimulus mean (V1 - V22).

Nowlis Mood Data

The Nowlis Mood Adjective Check List (MACL), Nowlis (1965), is a four point forced compliance check list, which requires subjects to rate their mood on 45 adjectives according to the degree to which the adjective applies to their present mood. The checks are:-

XX definitely yes

X yes

? do not know

NO definitely not

and were scored as 4, 3, 2, 1, respectively. The 45 adjectives break down into 12 mood dimensions by summing the scores for the relevant adjectives. The MACL was given prior to testing, at the end of the pretreatment phase, and at the completion of the testing phase to afford a measure of changes in mood state across the experimental session. The mood data were scored according to the dimensions suggested by Nowlis (1965), and expressed as both raw scores for each occasion and as change scores between occasions.

Analysis of variance was carried out on the raw scores and on the change scores, and the results are given in Table 10. The distribution of the data did not justify any transformation, but the scores are under the constraint of being derived from a forced compliance questionnaire that affords discrete scores as opposed to scores from a continuous scale. This implies a limitation on the degree of change because the subject's mood may have changed as a result of the experimental session, but the subject may not judge the mood change to be sufficient to move from one

class to the next.

There are fairly clear cut mood changes between groups that are evident from the histograms of the change scores, but these changes typically do not reach statistical significance. Further analyses were carried out to investigate sex and condition differences on the raw scores and on the change scores within each occasion. This provides more information about the differences than the H.O & P.O interaction effects which assess condition and sex effects globally over the three occasions and so attenuate any quadratic functions. The results will be discussed in terms of the two sets of change scores.

1. Pretreatment To Post-treatment Changes: The main findings of interest are that the escape group shows the largest decrease in anxiety and decrease in concentration. The non-escape group show a decrease in nonchalance whilst the escape group show an increase. The non-escape group also show the largest increase in depression and the largest decrease in pleasantness. The passive control group show the largest decrease in activation and the largest increase in deactivation.

2. Post-treatment to Post-test Changes: These changes are largely a recovery from the changes produced by the pretreatment phase. That is, the non-escape group became less depressed and more pleasant. Also evident are changes in the passive control group who drop in social affection, and deactivation together with an increase in activation.

To summarize these findings, it is clear that all subjects start off by reporting high anxiety which decreases over the experiment. The largest decrease observed was in the escape group. The non-escape group reported greater depression and less pleasantness after the pretreatment phase which recover after the test task. The other striking difference is the large increase in deactivation shown by the passive control group after the pretreatment phase which decreases after the test task. The analysis of these results reveal largely non-significant results save for the depression and nonchalance variables. The only sex effects of any note are a greater decrease in deactivation and social affection by the males, a greater decrease in aggression by the females, which occurs during the test task phase. There is also a larger increase in deactivation by the female subjects during the pretreatment phase.

To conclude, it seems that the mood data shows that the groups do differ in the way that the experimental contingencies affected the subjects. The non-escape subjects found the pretreatment more stressful compared to the escape group and the passive control group became most deactivated by this phase.

KEY 6. ITEMS ON THE NOWLIS AFFECT CHECK LIST

V1	AGGRESSION
V2	ANXIETY
V3	CONCENTRATION
V4	SOCIAL AFFECTION
V5	NONCHALANCE
V6	DEPRESSION
V7	PLEASANTNESS
V8	ACTIVATION
V9	DEACTIVATION
V10	STARTLED
V11	SCEPTICISM
V12	EGOISM
H	EXPERIMENTAL CONDITION
P	SEX
O	OCCASION THAT CHECK LIST WAS GIVEN

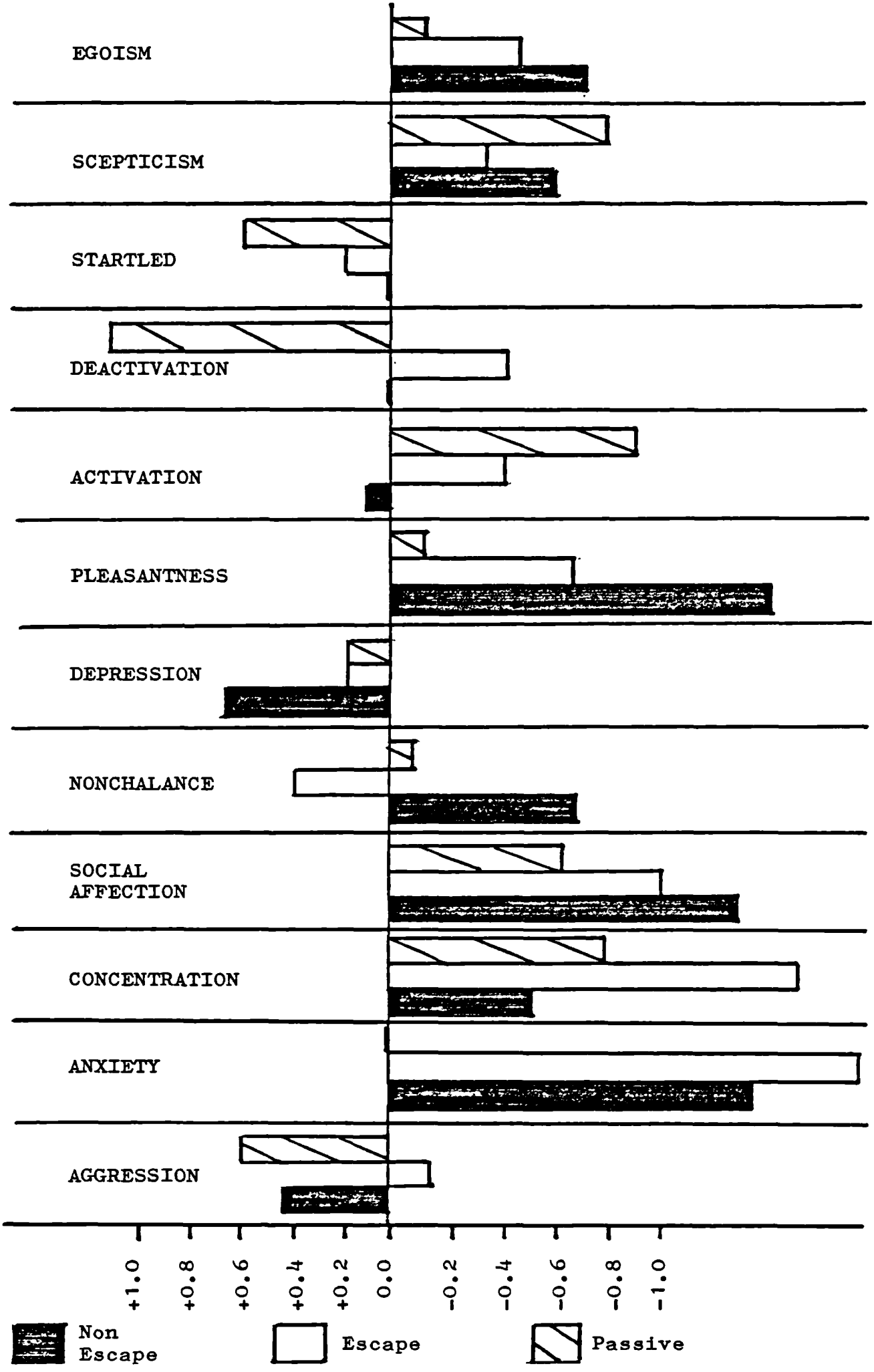
TABLE 10. ANALYSIS OF NOWLIS AFFECT CHECK LIST

	V1	V2	V3	V4	V5	V6
H	2.84	0.14	5.79	0.45	3.63	0.06
(2,24)	<.1	N.S.	<.01	N.S.	<.05	N.S.
P	1.48	0.81	3.43	2.51	0.15	0.23
(1,24)	N.S.	N.S.	<.1	N.S.	N.S.	N.S.
H.P	3.62	0.04	1.26	0.17	0.65	0.35
(2,24)	<.05	N.S.	N.S.	N.S.	N.S.	N.S.
O	0.79	10.46	2.56	8.81	3.88	0.81
(2,48)	N.S.	<.001	<.1	<.001	<.05	N.S.
H.O	0.44	0.77	0.35	0.60	1.86	2.46
(4.48)	N.S.	N.S.	N.S.	N.S.	N.S.	<.1
P.O	0.77	0.07	1.17	1.92	0.33	0.71
(2,48)	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
H.P.O	0.57	0.60	0.34	1.90	0.48	1.12
(4,48)	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

	V7	V8	V9	V10	V11	V12
H	3.71	3.46	4.04	0.15	0.39	2.34
(2,24)	<.05	<.05	<.05	N.S.	N.S.	N.S.
P	0.75	6.01	0.90	0.29	0.17	0.08
(1,24)	N.S.	<.025	N.S.	N.S.	N.S.	N.S.
H.P	1.50	1.95	0.18	0.50	1.13	1.65
(2,24)	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
O	2.19	2.11	2.48	1.92	2.13	0.61
(2,48)	N.S.	N.S.	<.1	N.S.	N.S.	N.S.
H.O	1.03	0.55	0.40	0.86	0.81	1.31
(4,48)	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
P.O	0.11	2.08	2.40	0.12	2.16	0.98
(2,48)	N.S.	N.S.	.1..	N.S.	N.S.	N.S.
H.P.O	1.64	1.00	1.04	0.82	0.95	0.18
(4,48)	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

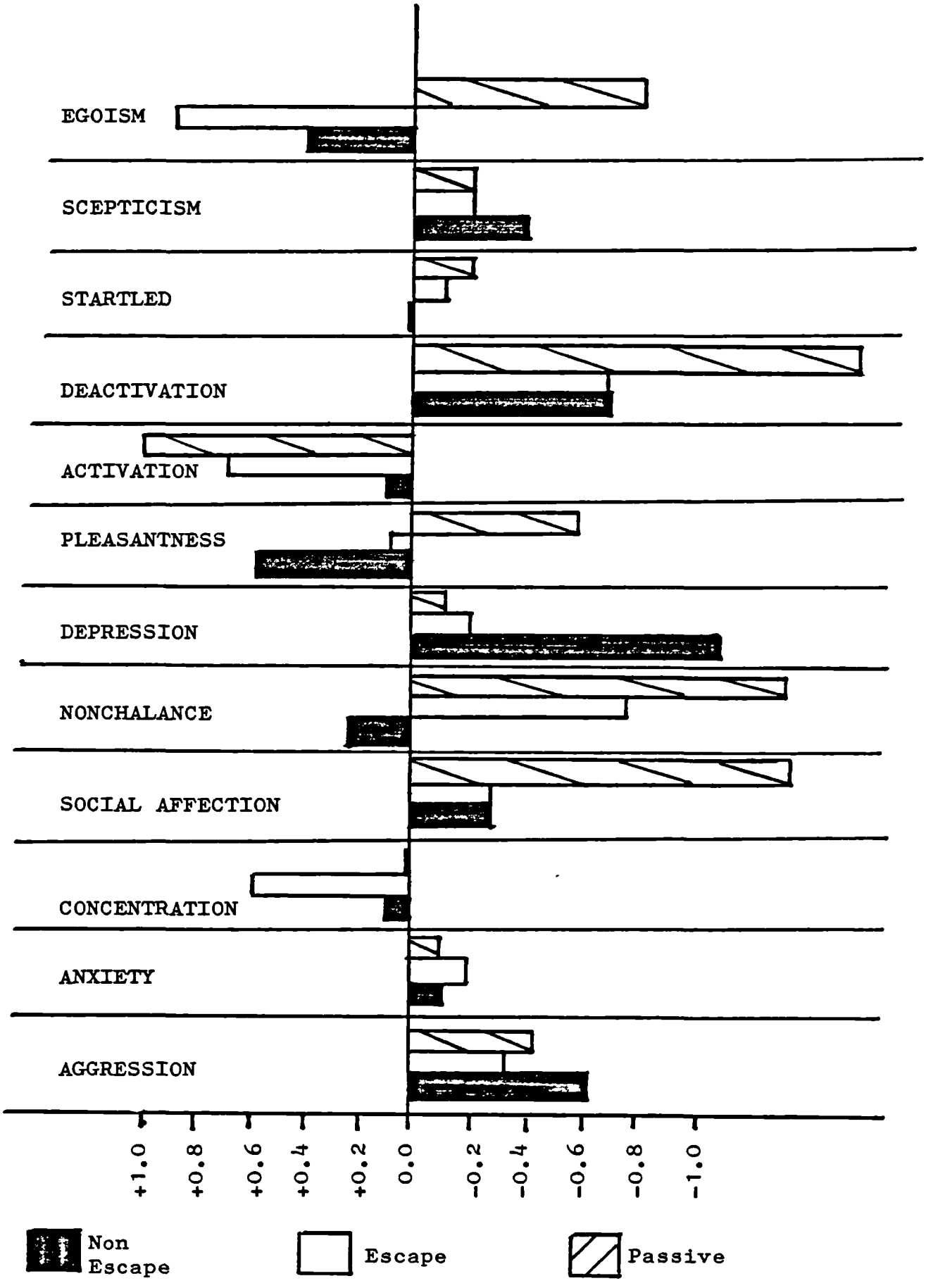
GRAPH 3. NOWLIS CHANGE SCORES (PRE-POST)

PRETREATMENT



GRAPH 4. NOWLIS CHANGE SCORES (PRE-POST)

TEST TASK



Miscellaneous Data

The data presented here were collected by means of questionnaire in order to look at trait individual differences and ratings of the experimental contingencies to which the subjects were exposed. Subjects were asked to rate their determination to succeed on the pretreatment phase of the experiment (Table 12), (the passive control groups are necessarily not included). All groups became less determined to succeed as the pretreatment phase proceeded - this is to be expected in the escape group where all subjects were successful after the first few trials. The drop in determination is more evident in the non-escape group and given that they fail on every trial this is in line with the predictions from learned helplessness theory in that exposure to uncontrollability will result in a motivational deficit. There is also a tendency for males to be less motivated than females. This drop in motivation reversed when S's were asked to rate their motivation to solve the anagrams (Table 13). The non-escape group were more highly motivated and the passive control group were least motivated. It may be expected that given consistent failure feedback on the first test, the relevant groups would be motivated to demonstrate competence on the anagram test. Also, the lower motivation of the passive control group may be a function of not being task involved in the pretreatment phase. These statements are obviously rather speculative.

The Mill Hill I.Q. scores are fairly homogeneous (Table 13) with the exception that males tend to score slightly higher than the females on this test.

Subjects were also asked to rate the noise on dimensions of unpleasantness and loudness (Table 11). It seems that the passive control groups found the noise more unpleasant and louder than the other two groups. Within the escape and non-escape groups there is a tendency to rate the noise as becoming more unpleasant and louder as the experiment progresses in the non-escape group. The opposite tendency occurs in the escape group. It should be noted, however, that these changes are relatively small.

The Eysenck Personality scores do not reflect any marked deviation from the population norms (Table 14) and also the age scores are fairly homogeneous, but this is to be expected as groups were selected so that age and personality would be equally represented across groups. Post hoc correlations are of limited value in assessing individual differences in relation to various aspects of the experiment as these data are based on n=5 for each group, and no analysis was attempted using these variables.

All subjects reported that they had believed the instructions concerning the aim of the experiment.

TABLE 11. RATINGS OF THE NOISE

UNPLEASANT	INITIALLY		FINALLY	
	MEAN	S.D.	MEAN	S.D.
G1	4.6	0.9	5.0	1.2
G2	4.6	0.9	4.2	0.4
G3	5.2	0.8	4.6	0.9
G4	3.8	0.4	3.6	1.5
G5	4.6	0.9	4.4	1.1
G6	5.2	0.4	5.2	1.1
H1	4.2	0.7	4.3	1.2
H2	4.6	0.9	4.3	0.8
H3	5.2	0.4	4.9	0.9
P1	4.8	0.8	4.6	0.8
P2	4.5	0.6	4.4	1.2

LOUDNESS	INITIALLY		FINALLY	
	MEAN	S.D.	MEAN	S.D.
G1	4.6	1.5	5.0	2.0
G2	4.2	0.8	3.8	1.1
G3	5.2	0.5	4.2	0.8
G4	4.0	0.7	4.2	1.9
G5	4.2	1.1	4.8	1.3
G6	5.2	0.8	5.0	0.7
H1	4.3	1.1	4.6	1.6
H2	4.2	1.1	4.3	1.1
H3	5.2	0.8	4.6	0.7
P1	4.6	0.9	4.3	1.3
P2	4.5	0.8	4.7	1.3

HIGH SCORE INDICATES GREATER LOUDNESS/UNPLEASANTNESS

TABLE 12. DETERMINATION TO SUCCEED ON PRETREATMENT

	INITIALLY		FINALLY	
	MEAN	S.D.	MEAN	S.D.
G1	2.8	1.7	4.0	2.3
G2	1.4	0.5	4.4	2.3
G3	-	-	-	-
G4	1.2	0.4	3.4	2.2
G5	2.4	1.7	2.8	1.8
G6	-	-	-	-
H1	2.0	1.2	3.7	2.1
H2	1.9	1.2	2.8	1.8
H3	-	-	-	-
P1	2.0	1.2	4.2	2.2
P2	1.8	1.2	3.1	1.9

TABLE 13. DETERMINATION TO SOLVE ANAGRAMS & MHIQ SCORES

	DETERMINATION		MHIQ	
	MEAN	S.D.	MEAN	S.D.
G1	2.4	1.5	120.0	6.6
G2	2.0	1.0	112.2	11.0
G3	2.0	0.7	122.2	6.9
G4	1.2	0.4	110.0	13.1
G5	2.4	1.7	117.2	11.7
G6	3.4	2.3	111.4	12.1
H1	1.8	1.1	115.0	9.8
H2	2.2	1.3	114.7	10.7
H3	2.7	1.6	116.7	9.3
P1	2.1	1.0	118.1	7.8
P2	2.3	1.5	112.8	11.4

LOW SCORE INDICATES GREATER DETERMINATION

TABLE 14. EYSENCK PERSONALITY SCORES AND SUBJECT AGE

	P		E	
	MEAN	S.D.	MEAN	S.D.
G1	4.4	2.7	6.6	5.7
G2	5.2	3.8	14.4	3.1
G3	2.4	2.4	13.6	4.4
G4	2.4	1.1	11.0	5.3
G5	3.6	1.5	15.4	2.6
G6	3.4	2.6	14.6	7.5
H1	3.4	1.9	8.8	5.2
H2	4.4	2.8	14.9	2.7
H3	2.9	2.4	14.1	5.8
P1	4.0	2.8	11.5	4.2
P2	3.1	1.7	13.7	5.1

	N		L	
	MEAN	S.D.	MEAN	S.D.
G1	8.2	4.7	7.0	3.5
G2	11.0	3.5	6.2	4.7
G3	9.4	4.3	2.8	3.3
G4	16.4	5.2	9.6	5.2
G5	19.4	2.9	7.0	4.9
G6	10.4	3.4	5.0	3.1
H1	12.3	4.8	8.3	4.2
H2	15.2	3.0	6.6	4.5
H3	9.9	3.7	3.9	3.0
P1	9.5	3.9	5.3	3.6
P2	15.4	3.7	7.2	4.2

	AGE	
	MEAN	S.D.
G1	38.6	14.3
G2	34.0	11.9
G3	33.4	14.3
G4	42.0	6.9
G5	36.6	11.2
G6	32.0	7.9
H1	40.3	10.6
H2	35.3	10.9
H3	32.7	10.9
P1	35.3	12.5
P2	36.9	8.3

DISCUSSION

Discussion

Based on the triadic design used by Hiroto & Seligman (1975), it was predicted that subjects exposed to uncontrollable noise would show certain deficits compared to subjects exposed to either controllable noise or noise that was passively tolerated. These deficits would be shown on an anagram solving test task and also result in physiological measures and self report measures indicative of the non-escape condition being more stressful. In general, the results are supportive of the distinction between controllable and uncontrollable stimulation but the group that passively tolerated the noise showed the greatest deficits on the test task. Before putting these findings into the context of the theory of learned helplessness, the results of the experiment will be reviewed.

The main dependent variables are the various measures of anagram solving performance. The results are mainly statistically nonsignificant, but the non-escape group show an inferior performance compared to the escape group. The passive control group have the poorest scores with the exception of the mean latency score computed for the first four anagrams. There are also clear cut sex differences on this test and it can be seen that females do worse than males.

The various measures correlate highly (+ ively) with each other indicating that they are not independent and also correlate negatively and significantly with the verbal I.Q. scores. Attempts to weight the anagram scores on the basis of the verbal I.Q. scores are not strictly justified

as the anagram scores are obtained after the pretreatment phase. The homogeneity of the I.Q. scores was such that when the scores were weighted the resulting analysis was virtually identical to the analysis on the untransformed scores. The results and analyses reported here are those carried out on these untransformed scores.

The skin conductance data show that the non-escape group showed an increase in basal level across trials compared to the other two groups which showed a decrease indicative of habituation. These divergent trends result in a crossover on Trial 8 between the escape and non-escape groups. The non-escape group also gave more skin conductance responses than the other two groups, with the passive control group showing the least responding. Assuming that these two measures are indices of stress, it seems that the non-escape group found the pretreatment phase more stressful than the other two groups. Males maintained a higher skin conductance level across trials and show more responding than females, but these differences do not change significantly over the various phases of the experiment and so are not of great interest in assessing experimental effects. The other dependent measures of response amplitude, rise time, and recovery time do not seem to be useful discriminants between experimental conditions.

The heart rate data are fairly extensive and without being unduly dismissive it seems that those results that reach statistical significance are largely due to the passive control males who maintain a higher tonic level and show greater variability in comparison to the other two groups. The other groups are fairly homogeneous and show a

gradual increase in tonic heart rate level across trials.

The mood data are more informative. Taking the change scores derived from the pre-experiment to the post-pretreatment phase, the non-escape group report becoming more depressed, less socially affectionate, less pleasant, less sceptical, and less nonchalant compared to the other group, whilst the escape group become less anxious, and the passive control group become most deactivated. The change scores obtained from the post-pretreatment phase to the post test task phase show that the non-escape group demonstrate the expected recovery in a decrease in depression and an increase in social affection and pleasantness. The escape group become slightly more depressed and the passive control groups become less socially affectionate and less deactivated.

Finally, the subjects' ratings of various aspects of the experiment indicate that the passive control group found the noise most unpleasant and loudest. The non-escape group found the noise increasing in aversiveness across trials, whilst the other groups rated a decrease in aversiveness across trials. The non-escape group were the most determined to solve the anagrams, and the passive control group were the least motivated to solve them.

With respect to the anagram scores - there are two major results that require explanation. Firstly, why it is that the passive control group shows the poorest performance. This is probably due to several factors. They were the least motivated group to solve the anagrams, they rated the noise as being more aversive than the other

groups which may have resulted in disruption on the test task due to the noise per se. Also, the passive control group are not task involved and are passively exposed to the noise so that their inferior performance may be due to this, indicated by their self reported increase in deactivation. It is also possible that this group is, in effect, also a non-escape condition and hence comparable to those subjects in the non-escape condition rather than the subjects in the escape condition. In order to test these hypotheses it would have been necessary to have included a control group that was merely left in the testing room for the appropriate length of time without being exposed to noise at all. This group was not included in the experimental design and so the following possibilities remain:-

a) deactivation due to lack of task involvement and together with perceiving the noise as being more aversiveness resulted in greater debilitation.

b) this group had become helpless as the subjects were exposed to contingencies similar to those that the non-escape group were exposed to.

c) chance selection of a group that were poor on anagram solving but the evidence from the I.Q. test scores does not support this.

The second finding that needs explanation concerns the fact that although there are differences between groups, these differences fail to reach statistical significance. There are two main possibilities for this, firstly, that the experimental manipulations were insufficient and so the results are attenuated or that even though the

experimental manipulations were sufficient the effects were attenuated because of a large amount of error variance. Given that this experiment involved the use of instructional set and non-veridical feedback to induce the perception of control or non-control, it is important to establish that the non-significance of the results is due to the inflated error variance. The skin conductance data indicate that the experimental conditions were sufficient to induce physiological discrimination between groups and the subjects' self reports indicate that the manipulations were effectively convincing. The Mill Hill I.Q. data, and the Eysenck personality data do not indicate any excessive heterogeneity on these variables in the subject sample. The standard deviations of the test task scores are comparable to those reported in the Hiroto & Seligman (1975) study, suggesting that it is not differences in anagram solving ability per se that have inflated the error variance. The subjects in this study are not college students, but are volunteers from the general public, from an unknown background of attitudes, education, etc. that may well affect the way that the subjects perceive the experimental test session. For example, a middle aged subject is probably less likely to be affected by the perception of non-control in an experiment against the background of his life experience, compared to a student subject who is in competition with his peer group in university.

These variables have been classified by Abramson et al (1978), as the attributional components of helplessness. That is, a subject will show learning deficits to the extent to which the perceived non-control is rationalised as being

due to a stable perception of a generalised lack of personal ability. If non-control is perceived as being due to external factors, or is specific to the pretreatment task, or is an unstable perception, then the learning deficits will be reduced or even non-existent. Assuming that these processes are important and relevant, then heterogeneity on these components will be maximized in the reported study by using volunteers from the general public.

Based on the self report data, the skin conductance data, and the anagram test task results it appears that the experiment is supportive of the theory of learned helplessness with two qualifications. Firstly, that the passive control group may be of different status experimentally than that for which it was intended. Secondly, that the test task results are attenuated due to uncontrolled individual differences in the perception and attribution of non-control.

CHAPTER 7

THE MAIN STUDY: LEARNED HELPLESSNESS - DEFICITS ASSOCIATED WITH NON-CONTROL OR FAILURE

INTRODUCTION

Introduction

The pilot study investigated the use of instructional set and non-veridical feedback as independent variables in the learned helplessness paradigm. The results were interpreted as being supportive of the theory of learned helplessness, but many of the measures taken failed to reach statistical significance. It was suggested that this was due to the heterogeneity of the subject sample. That is, subjects were from a wide age range and from different backgrounds together with the bare minimum of information about the experiment, that is, they were told what was required of them and not given a complete account of what the tests were about. It is possible that these factors contributed to a variety of possible interpretations of the experimental session which resulted in an inflated error variance on the measures taken.

The main study to be reported was an extension of the pilot study. The subject sample selected was more homogeneous and attempts were made to manipulate expectations and attributions about the experimental session as independent variables.

The literature review has covered the development of the theory of learned helplessness and cited evidence in support of the theory. An account of the reformulation of the theory has been given and as this is of direct relevance to the main study this reformulation will be summarized. The reformulation proposes that the deficits are not due to exposure to uncontrollability per se, but to the perception of uncontrollability with an appropriate attribution relating to this perception. The attributions are formalised

as follows:-

1) GLOBAL - SPECIFIC : relates to generalisation
2) STABLE - UNSTABLE : relates to the time course

} of the helplessness deficits.

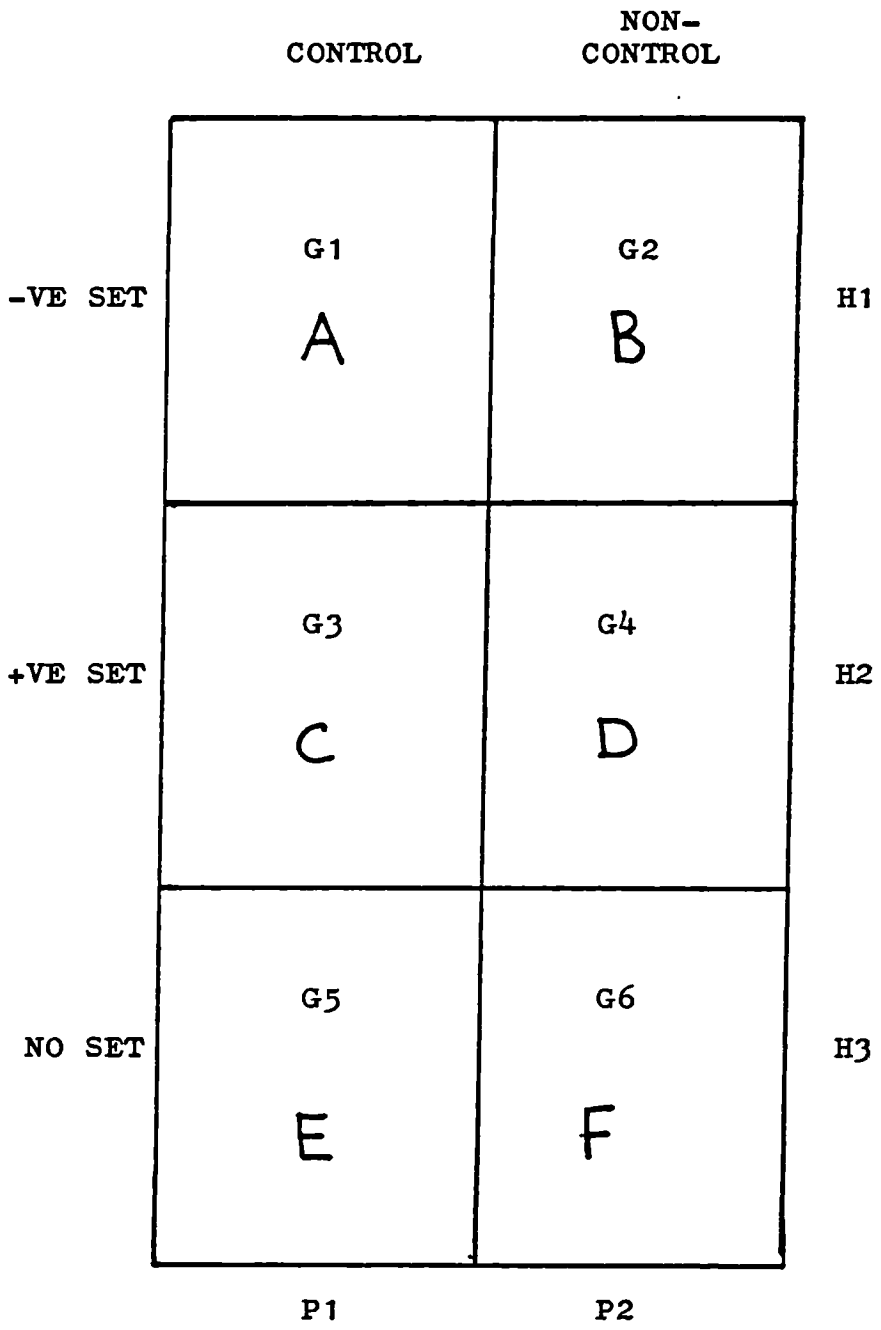
3) INTERNAL - EXTERNAL: relates to the perception of response outcome independence (PROI).

Given these processes, a subject in a helplessness type experiment will show deficits on a test task after uncontrollable pretreatment if he believes that his previous performance was due to a stable perception of his lack of general ability. If a subject believes that his performance was due to bad luck, was due to his lack of ability on that specific task only, or if his perception is unstable then the deficits will be attenuated.

The criticisms of this reformulation have already been given in a previous chapter and an alternative position has been proposed. This alternative will now be summarized briefly.

All of the human experiments involve the inherent component of success or failure whenever subjects are set to control stimuli in an experimental task. That is, does the subject who is unable to control the stimulus perceive response-outcome independence or does he perceive failure on a psychology test ? Are the deficits due to proactive interference as a function of response-outcome independence or due to dysphoria as a function of the perception of failure ? It is necessary to ensure that perception of failure and the perception of response-outcome independence are not synonymous, and given the reformulation of the

FIGURE 10. FIGURE SHOWING EXPERIMENTAL DESIGN OF MAIN STUDY



G n=12
H n=24
P n=36

basic theory this is not easy to state explicitly. The distinction lies in how the experimental subject evaluates the experience. That is, given that the subject has been unable to control the stimulus in the experiment he will perceive this as failure to the extent to which he believes that the task can be successfully done by other subjects. If the subject believes that no one can be successful, he is unlikely to perceive his performance in terms of failure, but in terms of the impossible demand of the experimenter. Contrariwise, if he believes that everyone else can successfully do the test when he is able to control the stimulus then he is unlikely to perceive the experience as success as it is essentially trivial. This distinction is made more explicit by considering the experimental design of the main study.

The experiment involves a 3 X 2 design (Figure 10). Subjects in the CONTROL block (cells A, C, E) receive feedback that they are controlling the aversive noise stimulation and subjects in the NON-CONTROL block receive feedback that they are not controlling the aversive noise stimulation. Crossed with these conditions are levels of cognitive set. Subjects in the +VE SET block (cells C, D) are instructed that the experiment is very difficult, most people are unable to do it and so, given the failure feedback they should not worry but try again. Given the success feedback they are doing extremely well, better than the majority of subjects and they should carry on. This condition minimises the perception of failure on the pretreatment.

Subjects in the -VE SET block (A, B) are instructed that the task is very easy and that given success they are only doing as well as most people and that they must try harder. Given the failure feedback then they are doing very poorly and must try a lot harder as most people can do the task. This condition maximises the perception of failure on the pretreatment. All subjects are shown the feedback slides and told that they can get any slide on any particular trial depending on their performance. Depending on the group to which they had been assigned they receive the feedback appropriate to that group on each trial.

The relevance of this is that success/failure is crossed with control/noncontrol. Subjects in cell A are told that they control but the set and feedback that they receive should induce the perception of failure. Also, subjects in cell D do not have control but their set and feedback should induce the perception of success.

All subjects are then tested on an anagram solving test task to assess any deficits in their performance. The major dependent variables are the anagram scores, physiological measures of skin conductance, heart rate, and respiration as indices of stress, and mood change inventories to assess mood changes. Manipulation checks are included in the post-experimental interview and state/trait components are assessed using the appropriate questionnaires.

The experimental design also includes two groups that receive minimal instructional set and plain feedback slides to act as controls for the set and feedback manipulations employed in the other groups. The analysis of the data

should allow the following comparisons (Figure 10):-

A) There are deficits associated with control/noncontrol and not with the level of set. Hence, the most important dimension is that of controllability, supportive to the learned helplessness theory.

B) There are deficits associated with the level of set and not with the level of control. So, it is the perception of success/failure that is the major determinant of the deficits.

The interaction terms provide considerable information. Given that both success/failure and the dimension of controllability are important then it might be predicted that the rank order of the deficits, by group, will be B, A, D, C. Also, that the cells E,F should show greater variability as the subjects in these groups are not given the context within which to interpret their performance.

This experiment allows a direct test of whether the deficits observed in the human experiments are a function of success/failure or a function of control/noncontrol.

METHOD

Method

Subjects

Subjects were 72 female volunteers from the general public by means of advertisements in the press or notices in local colleges. Twelve subjects were randomly assigned to one of six experimental groups. Data relating to I.Q. and personality are cited in the appendix. All subjects received payment for their participation in the experiment. Female subjects were chosen for this experiment in order to avoid a sex effect and based on the first study their mood changes were consistent with predictions from the theory of learned helplessness. That is, female subjects self reported feelings of depression whilst male subjects tended to self report aggression. There were also considerable sex differences on the measure of skin conductance onset basal level.

Apparatus

The pretreatment phase of the experiment required subjects to attempt to control noise (95 decibel) presented over a pair of headphones from a tape recorder. The noise was synthesised on a Synthi VC synthesiser and recorded onto magnetic tape. The control box measured 3" X 1" and consisted of a single pushbutton. The subject held the control box in her dominant hand and was instructed to press the button with her thumb only, in order to minimise bodily movement. Subjects were provided with trial by trial feedback on slides which were back projected onto a screen facing the subject. The slides were red for the

non control feedback and green for the control feedback, with lettering appropriate to the condition to which the subject had been assigned, giving information about success or failure.

The test phase of the experiment consisted of a series of 10 soluble five letter anagrams selected by Hiroto & Seligman (1975) from the Tresselt & Mayzner (1966) list. The anagrams were presented on slides by means of a Kodak carousel slide projector, and were back projected onto the screen facing the subject. The slide projector was operated by a second experimenter who was seated in the testing room with the subject, and who also timed the subject's anagram solving performance with a stopwatch. It should be noted that the pretreatment phase of this experiment consisted of only 10 trials and that only 10 anagrams were presented. This reduction in the number of trials was used in order to minimise subjects' suspicions which might be aroused by the repeated presentation of the same feedback slide, that is, subjects typically expect to do better as the experiment progresses. The number of anagrams was reduced in order to cut down on the testing time and also because it was found in the first study that any deficits observed occurred most evidently during the first four trials.

Heart rate, skin resistance, and respiration were recorded throughout the entire experiment. Silver/silver chloride electrodes were attached to the ventral sides of the subject's first and second fingers of the non-preferred hand to record skin resistance. The electrodes were checked prior to use and if they were found to have a

resistance of 2 Kilohms or more they were re-chlorided. The use of plastic masks with 1 cm holes (corresponding to the diameter of the electrodes) ensured that the recording was kept constant for each subject. An isotonic saline gel was used as a contact medium. Heart rate was measured by means of stainless steel plate electrodes attached to each ankle and the wrist of the non-preferred hand of the subject. The recording sites were abraded with sand paper before the application of hypertonic saline gel which was used as a contact medium. Respiration was recorded by means of a bead thermistor placed under one of the subject's nostrils. These physiological measures were recorded on paper chart via an Elema Schonander Mingograph and also onto magnetic tape via a Hewlett Packard Instrumentation recorder. The presentation of the stimuli was controlled by digitimers triggered from a pre-recorded magnetic tape.

Procedure

Subjects were randomly assigned to one of six experimental groups. The details of these groups are as follows:-

The experimental design was a 3 X 2 design which involved the dimension of control/non-control crossed with three levels of cognitive set. Subjects in the control condition were presented with green feedback slides at the end of each trial which signified successful control over the noise stimulus. The non-control subjects were presented with red feedback slides at the end of each trial signifying that they had failed to control the noise stimulus.

The levels of cognitive set were manipulated using

both the instructions and the lettering on the feedback slides. The details of these sets are:-

A) -ve set: Subjects in this condition were instructed that the experiment was very easy and that most subjects were able to reach the highest level of feedback. These subjects received ten presentations of the same slide at the end of each trial and irrespective of whether they had controlled the noise or not were set to evaluate their performance negatively.

B) +ve set: Subjects in this condition were instructed that the experiment was very difficult and that most subjects were unable to reach the higher levels of feedback. These subjects also received the same feedback slide at the end of each trial and similarly were set to evaluate their performance positively irrespective of whether they had been able to control the noise or not.

C) no set: Subjects in this condition were given the bare minimum of instructions and were given no information as to how well they could expect to do. The feedback slides were plain red or green slides.

The exact instructions used and the slides are shown in the appendix. All subjects were unable to control the noise stimuli but were given feedback to induce the perception of control or non-control.

All subjects were seated in a comfortable chair and given a 5 second sample of the noise. Recording electrodes were then attached and the subject was asked to complete the following questionnaires:-

a) Eysenck Personality Questionnaire

b) Rotter Locus Of Control Questionnaire

c) Beck Depression Inventory

d) Mill Hill test of verbal I.Q.

e) Lader Affect Check List

Subjects were then given instructions appropriate to the group to which they had been assigned and left in the testing room for a five minute rest period. Ten unsignalled bursts of noise were administered, which were identical in duration and inter-trial interval (mean duration = 10 seconds, mean ITI = 60 seconds). Subjects were required to press the pushbutton as many times as possible whilst the noise was on in order to turn it off. At the end of this phase the experimenter entered the room and gave the subject another Lader Affect Check List to complete. The subject was then requested to stay on and do another test for someone else in the department. All subjects agreed to do so and were then introduced to the second experimenter who gave the subject a written set of instructions. The anagram test task was then presented by the second experimenter who was seated behind the subject in the test room. Time to solution was recorded for each anagram using a stopwatch and was noted onto a score sheet. If the subject failed to give the correct solution within 100 seconds the next anagram was presented.

After the anagram test task the first experimenter returned and gave the subject another Lader Affect Check List to complete. The subject was then asked to complete a post-experimental questionnaire relating to attributions

for their performance on the tests, ratings of the noise, motivation to do the tests etc.

All subjects were fully debriefed and paid.

RESULTS

Anagram Results

The anagram test task is the major dependent variable in the experimental studies as it measures the extent to which the pretreatment phase of the experiment affects the performance of the subject on a subsequent unrelated task. As in the first study, a variety of measures can be obtained from the raw data, but these are not necessarily independent as the correlation matrix in Table 16 shows. The raw data are tabulated in Table 15 and the data were transformed where appropriate prior to analysis. The results from the analyses of variance are given at the foot of the page of the results tables.

The results from this study are relatively clear cut. For all variables (with the exception of CP - the conditional probability of solution), there is a significant group effect (G). There is a significant effect for the dimension of control/non-control (P). The only variable for which the dimension of cognitive set approaches significance is TC - trials to criterion. There are no significant differences for this effect on the other variables. It is also evident that none of the interaction terms between level of cognitive set and controllability (H X P), reaches statistical significance.

The evidence suggests that the dimension of controllability (P) is the most important determinant of the deficits on the test task, but it should be noted that the negative set non-control group (G2) showed the worst performance compared to all other groups. Whilst the interaction terms failed to reach statistical significance, it can be seen that there is an effect when non-control is

presented in the context of negative cognitive set. That is, these dimensions interact behaviourally in this combination but not in other combinations of control and cognitive set.

All subjects were asked during the post experimental interview whether it was more important to them that they had succeeded/failed on a psychology test or whether it was more important that they had been able/unable to control the noise stimulus. The results from this question are given in Table 17, and it can be seen that there are no significant differences in the frequencies of the subjects reporting success/failure or control/non-control between G, H and P. However, for the levels of cognitive set H1 (which attempted to maximise the perception of failure) and H2 (which attempted to minimise the perception of failure and maximise the perception of success) more subjects reported the success/failure component as being more important. There are no differences on this between levels of controllability (P).

The data were re-analysed comparing these subjective perceptions as an effect and there were no significant differences on any of the variables for this. However, the H X FC (set X self report perception of failure or control) interaction for the number of failures reaches statistical significance ($p<.005$) as does the H X FC interaction for the conditional probability of solution ($p<.005$). All other interaction terms are non-significant. The interaction for the failures indicates that the subjects that reported that the perception of failure was most important in conditions H1 (-ve set) and H3 (no set) had

KEY 7. KEY TO ANAGRAM RESULTS

G1	-VE SET CONTROL
G2	-VE SET NON-CONTROL
G3	+VE SET CONTROL
G4	+VE SET NON-CONTROL
G5	NO SET CONTROL
G6	NO SET NON-CONTROL
H1	-VE SET
H2	+VE SET
H3	NO SET
P1	CONTROL
P2	NON-CONTROL

TABLE 15. ANAGRAM RESULTS

	MLS 1		MLS 2		FAILURES	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
G1	12.45	9.36	18.36	13.45	0.75	0.86
G2	25.10	7.92	41.73	17.03	2.25	1.86
G3	13.40	9.14	19.28	15.12	0.75	1.06
G4	18.43	11.27	36.80	27.28	2.33	2.84
G5	14.51	11.98	22.21	13.58	0.92	0.90
G6	19.98	12.23	32.06	21.55	1.67	1.78
H1	18.78	10.66	30.20	19.07	1.50	1.62
H2	15.91	10.36	28.04	23.52	1.54	2.25
H3	17.21	12.12	27.13	18.32	1.29	1.43
P1	13.45	9.95	20.06	13.75	0.81	0.92
P2	21.15	10.73	36.86	22.19	2.08	2.17

	SOURCE	F	d.f.	p
MLS 1	G	2.63	5,66	<.05
	H	0.36	2,66	N.S.
	P	10.23	1,66	<.005
	H.P	1.11	2,66	N.S.
MLS 2	G	2.52	5,66	<.05
	H	0.16	2,66	N.S.
	P	10.34	1,66	<.005
	H.P	0.97	2,66	N.S.
FAILURES	G	2.26	5,66	<.10
	H	0.13	2,66	N.S.
	P	9.94	1,66	<.005
	H.P	0.44	2,66	N.S.

TABLE 15. ANAGRAM RESULTS (contd.)

	TRIALS TO CRITERION (TC.)		CONDITIONAL PROBABILITY OF SOLUTION (CP.)	
	MEAN	S.D.	MEAN	S.D.
G1	6.42	2.35	0.924	0.09
G2	8.92	1.88	0.766	0.19
G3	5.50	2.71	0.912	0.12
G4	6.83	2.72	0.763	0.31
G5	6.33	1.97	0.872	0.13
G6	7.42	2.15	0.811	0.21
H1	7.67	2.44	0.849	0.16
H2	6.17	2.75	0.837	0.24
H3	6.88	2.09	0.842	0.17
P1	6.08	2.33	0.902	0.11
P2	7.72	2.39	0.783	0.24

	SOURCE	F	d.f.	p
TC	G	3.05	5,66	<.025
	H	2.50	2,66	<.10
	P	8.96	1,66	<.005
	H.P	0.64	2,66	N.S.
CP	G	1.60	5,66	N.S.
	H	0.03	2,66	N.S.
	P	7.13	1,66	<.001
	H.P	0.42	2,66	N.S.

TABLE 16. INTERCORRELATION MATRIX OF ANAGRAM SCORES

	MLS 1	MLS 2	FAILURES	TC	CP
MLS 1	1.00				
MLS 2	0.732**	1.00			
FAILURES	0.410**	0.915**	1.00		
TRIALS TO CRITERION	0.724**	0.763**	0.591**	1.00	
CONDITIONAL PROBABILITY	-0.335*	-0.835**	-0.926**	-0.539**	1.00

** $p < .001$

* $p < .002$

TABLE 17. FREQUENCY OF SUBJECTS REPORTING SUCCESS/FAILURE OR CONTROL/NON-CONTROL AS MOST SALIENT IN THE EXPERIMENT.

	CONTROL/NON-CONTROL	SUCCESS/FAILURE
G1	6	6
G2	5	7
G3	3	9
G4	7	5
G5	9	3
G6	7	5
H1	11	13
H2	10	14
H3	16	8
P1	18	18
P2	19	17

had more failures than their counterparts that reported control as the most important aspect of the experiment. The reverse is true for subjects in the H2 (+ve set) condition. The conditional probability interaction is essentially the same with the self report failure subjects showing a lower conditional probability of solution than the self report control subjects in conditions H1 and H3. Again, the reverse is true for those subjects in the H2 condition.

This indicates that the perception of failure within the negative set and no set groups contributes to deficits on the anagram task and that the positive set of condition H2 attenuates these deficits in the failure subjects. However this is not evident in the other variables derived from the anagram data so the generality of this is questionnable. Referring to the tables of means for this breakdown (Table 18) it appears that those subjects that self report failure saliency perform worse on the anagram task for all groups in the negative and no set conditions (i.e. G 1, 2, 5, 6). This is not so clear in the positive set groups (i.e. G 3, 4). Also, this observation is not supported by statistical significance in the relevant analyses.

It was expected that G3 (the positive set control group) would perform better than any other group on the anagram test task and yet they do not do as well as G1 (negative set control group) on the latency measures and their superiority on other measures is marginal. Referring to the miscellaneous data (Table 35), it can be seen that this group has a lower score on the Rotter Locus Of Control

Questionnaire than the other groups (i.e. they are more internal), and hence may not be influenced so much by environmental contingencies and so the cognitive set may have had little effect. There is a positive correlation between externality on the Rotter and the anagram scores. That is, the greater the score on the Rotter the poorer the anagram solving performance, but this correlation does not reach statistical significance. This group also scores highly on the P scale of the Eysenck Personality Questionnaire. That is this group is more 'tough minded' than the other groups which also may have attenuated the group's performance. The correlation between P and anagram solving performance is variable in direction and non-significant. An alternative explanation is that this group had reached a ceiling for anagram solving and so any positive feedback/ set would have little effect.

To summarize the anagram results, it is clear that the dimension of control/non-control is the major determinant of performance on the anagram solving test task. The levels of cognitive set alone do not affect the anagram scores, nor do the interactions between levels of set and control/ non-control reach significance. However, the poor performance of the negative set non-control and the evidence from the breakdown based on the subjects' perception of the experiment indicate that the perception of failure is an important component of the anagram results and that this perception was, to some extent, affected by the levels of cognitive set employed.

TABLE 18. BREAKDOWN OF ANAGRAM SCORES BY SUBJECT'S PERCEPTION OF SUCCESS/FAILURE OR CONTROL/NON-CONTROL

		MLS1		MLS2	
		MEAN	S.D.	MEAN	S.D.
G1	F	14.73	11.93	25.75	14.75
	C	10.16	6.14	11.61	7.78
G2	F	24.64	10.02	43.61	21.98
	C	25.73	4.58	39.08	7.59
G3	F	15.06	10.08	20.53	17.31
	C	10.06	6.84	16.78	11.22
G4	F	16.91	9.70	22.98	15.61
	C	19.51	12.90	46.65	30.98
G5	F	20.58	22.16	33.26	21.73
	C	12.49	7.13	18.52	8.62
G6	F	26.92	15.46	48.18	21.41
	C	14.89	6.66	20.54	13.22
H1	F	20.07	11.66	35.36	20.45
	C	17.24	9.66	24.10	16.09
H2	F	15.77	9.57	21.48	16.05
	C	16.07	11.70	35.79	28.99
H3	F	24.55	16.96	42.59	21.36
	C	13.54	6.91	19.41	10.51
P1	F	15.92	12.46	24.62	16.76
	C	11.24	6.59	15.97	8.97
P2	F	23.04	11.77	38.89	21.74
	C	19.45	9.69	35.04	23.02

TABLE 18 (contd.)

		FAILURES		TC	
		MEAN	S.D.	MEAN	S.D.
G1	F	1.33	0.82	7.00	2.53
	C	0.16	0.41	5.83	2.23
G2	F	2.57	2.37	8.14	2.19
	C	1.80	0.84	10.00	0.00
G3	F	0.75	1.17	6.50	2.83
	C	0.75	0.95	3.50	0.58
G4	F	0.80	0.84	6.80	2.68
	C	3.43	3.31	6.85	2.97
G5	F	1.67	0.58	7.33	3.06
	C	0.67	0.87	6.00	1.58
G6	F	3.00	1.87	8.80	1.79
	C	0.71	0.95	6.42	1.90
H1	F	2.00	1.87	7.62	2.33
	C	0.91	1.04	7.72	2.69
H2	F	0.77	1.01	6.62	2.66
	C	2.45	2.94	5.64	2.87
H3	F	2.50	1.60	8.25	2.25
	C	0.69	0.87	6.18	1.68
P1	F	1.11	0.99	6.82	2.60
	C	0.53	0.77	5.42	1.89
P2	F	2.18	2.00	7.94	2.25
	C	2.00	2.35	7.53	2.55

TABLE 18 (contd.)

		CP	
		MEAN	S.D.
G1	F	0.87	0.09
	C	0.98	0.05
G2	F	0.76	0.25
	C	0.80	0.08
G3	F	0.92	0.12
	C	0.90	0.14
G4	F	0.93	0.07
	C	0.65	0.37
G5	F	0.75	0.03
	C	0.91	0.12
G6	F	0.67	0.29
	C	0.91	0.12
H1	F	0.81	0.19
	C	0.90	0.11
H2	F	0.92	0.10
	C	0.74	0.32
H3	F	0.70	0.19
	C	0.91	0.11
P1	F	0.87	0.12
	C	0.93	0.10
P2	F	0.78	0.22
	C	0.79	0.26

Lader Affect Check List Results

The Mood Affect Check Lists were scored by measuring, in millimetres, the distance from the margin on the left hand side of the questionnaire to the subject's check on that dimension. The questionnaire was given on three occasions - 1) pre-experiment 2) post pretreatment 3) post experimentally. The data analysed were the pre-experimental scores in order to assess whether there were any group differences on the mood dimensions prior to the experimental intervention, and the two sets of change scores derived from the three occasions. The variables are as follows:-

X - pre-experimental scores

Y - post pretreatment scores

Z - post experimental scores

C - (Y - X)

D - (Z - Y)

Frequency distributions of the scores (both raw scores and change scores), revealed an adequate normal distribution of scores which did not justify a transformation prior to analysis. Analysis of variance was carried out on these sets of scores. The results from the analyses are given in Table 19. Key to the Analysis is Key 8.

The only dimension for which differences are evident pre-experimentally was ATTENTIVE/DREAMY where there was a significant difference ($p<.05$) between P1 and P2 (control/non-control) with the P2 non-control group reporting to be more 'dreamy'. Analysis of the change scores between phases of the experiment showed the following:-

1) Mood changes resulting from the pretreatment phase were that non-control subjects (P2) reported greater feebleness, more clumsiness, slowness, greater incompetence, more sadness, and being more withdrawn. The H1 subjects (negative set), reported becoming muzzier, clumsier, more discontented, and more incompetent.

2) Mood changes following the test task were that non-control subjects (P2), reported being less clumsy, discontented, and less incompetent. The negative set subjects (H1) reported being less discontented and less incompetent, and also more relaxed. The significant interactions show that G2 (negative set non-control), reported being calmer and more relaxed.

It can be seen that the mood changes occurring from the end of the pretreatment phase to the end of the test phases are largely recovery from the mood changes brought about by the pretreatment phase. These findings are in line with the predictions from the theory in that the non-control subjects reported mood changes congruent with the debilitation contingent upon such stimulation. The results also show that the H1 condition subjects were distinct from the other two levels of cognitive set in that they reported being more affected by the experience of uncontrollability. These data support the theory of learned helplessness and also add strength to the conclusion that the experimental conditions were effective.

Further analysis reveals that the experimental conditions of instructional set did directly affect the mood of the subject. Comparing each set manipulation against the appropriate no set condition for both control and non-control conditions, the following is clear. The no set control group (G5) self report being less clumsy and more proficient after the controllable pretreatment. Compared to this, the negative set control group (G1) self report being more muzzy, slower, more antagonistic, and not less clumsy and not more proficient. The positive set control group (G3) self report being more excited and more attentive relative to G5. So, even for conditions where control is available to the subject the instructional set condition does affect the subjects self report. The effects are even more apparent when we consider the non-control conditions. The no set non-control group (G6) self report being feebler, more clumsy, slower, less proficient, and more antagonistic. As well as these changes the group which received negative set in the non-control condition (G2) also reported being more excited, more muzzy, more discontent, more troubled, more tense, more sad, but not more slow. The positive set non-control subjects (G4) self report being more sad, and more antagonistic - none of the other mood changes found in the no set and negative set conditions were to be found.

All these mood changes reported here are significant at the .05 level or less.

So, it is clear that the experimental manipulation of instructional set as well as being defined operationally has had a behavioural effect on the subjects. In general the negative set produces negative mood changes, and the positive set tends to alleviate those negative mood changes which result from the no set non-control condition.

These results confirm the efficacy of the manipulation and the validity of the experimental design.

KEY 8. KEY TO LADER AFFECT CHECK LIST ANALYSIS

	LOW	HIGH
V1	ALERT	DROWSY
V2	CALM	EXCITED
V3	STRONG	FEEBLE
V4	MUZZY	CLEAR
V5	WELL CO-ORDINATED	CLUMSY
V6	LETHARGIC	ENERGETIC
V7	CONTENDED	DISCONTENTED
V8	TROUBLED	TRANQUIL
V9	SLOW	QUICK WITTED
V10	TENSE	RELAXED
V11	ATTENTIVE	DREAMY
V12	INCOMPETENT	PROFICIENT
V13	HAPPY	SAD
V14	ANTAGONISTIC	AMICABLE
V15	INTERESTED	BORED
V16	WITHDRAWN	GREGARIOUS

X PREEXPERIMENTAL SCORE

Y POST PRETREATMENT SCORE

Z POST TEST SCORE

C CHANGE SCORE (Y - X)

D CHANGE SCORE (Z - Y)

TABLE 19. ANALYSIS OF LADER AFFECT CHECK LIST DATA

		H	P	H X P
df		(2,65)	(1,65)	(2,65)
ALERT/	X	0.140	0.107	0.093
DROWSY	C	1.934	0.032	0.018
	D	0.191	0.301	0.001
CALM/	X	0.304	0.614	0.113
EXCITED	C	0.532	0.038	2.614*
	D	1.736	0.973	4.203*
STRONG/	X	1.091	0.485	0.569
FEEBLE	C	2.430	13.792****	0.541
	D	0.447	3.986*	1.977
MUZZY/	X	0.407	3.098	2.574
CLEAR	C	4.710*	0.002	1.552
	D	1.031	0.001	0.718
WELL COORD/	X	1.782	1.261	0.576
CLUMSY	C	3.985*	12.853****	3.042
	D	0.515	8.430***	2.438
LETHARGIC/	X	0.919	0.617	0.232
ENERGETIC	C	1.988	0.610	0.297
	D	0.387	0.022	0.074
CONTENTED/	X	1.141	3.800	1.562
DISCONTENTED	C	3.815*	2.503	0.352
	D	3.965*	5.189*	1.582
TROUBLED/	X	0.438	0.079	0.877
TRANQUIL	C	1.982	3.547	0.548
	D	3.007	1.181	2.721
SLOW/QUICK	X	1.230	2.593	0.105
WITTED	C	1.504	7.582**	1.738
	D	0.421	1.566	1.498
TENSE/	X	0.443	0.090	0.082
RELAXED	C	1.080	0.283	1.923
	D	4.314*	0.000	4.952**

* <.05
** <.01
*** <.005
**** <.001

TABLE 19. (contd.)

		H	P	H X P
df		(2,65)	(1.65)	(2,65)
ATTENTIVE/ DREAMY	X	0.078	3.982*	1.153
	C	0.066	0.692	2.712
	D	0.445	0.003	1.557
INCOMPETENT/ PROFICIENT	X	0.633	1.392	0.480
	C	4.549*	27.095****	2.375
	D	3.322*	9.185***	1.426
HAPPY/ SAD	X	0.137	0.125	1.695
	C	0.770	11.814****	0.384
	D	0.975	2.512	0.743
ANTAGONISTIC/ AMICABLE	X	2.819	0.317	0.875
	C	2.403	3.865	1.022
	D	0.794	0.865	0.863
INTERESTED/ BORED	X	0.324	0.221	0.684
	C	2.470	0.744	1.263
	D	1.604	0.746	0.241
WITHDRAWN/ GREGARIOUS	X	0.454	0.040	1.859
	C	0.054	4.299*	1.189
	D	2.700	0.150	0.234

* <.05
** <.01
*** <.005
**** <.001

TABLE 19 (A)

TABLE OF SIGNIFICANCE LEVELS FOR THE INDIVIDUAL GROUP ANALYSIS OF LADER AFFECT CHECK LIST DATA COMPARING BEFORE AND AFTER PRETREATMENT SCORES

	G1	G2	G3	G4	G5	G6
V 1	.657	.201	.223	.060	.124	.581
V 2	.484	*.008	*.018	.623	.888	.261
V 3	.674	*.038	.135	.404	.221	*.006
V 4	*.007	*.050	*.026	.953	.822	.369
V 5	.107	*.033	.350	.091	*.000	*.005
V 6	.196	.180	.598	.198	.221	.933
V 7	.078	*.001	.880	.731	.325	.249
V 8	.162	*.006	.324	.272	.151	.371
V 9	*.021	.178	.498	.169	.104	*.004
V10	.484	*.003	.148	.809	.858	.485
V11	.087	.839	.086	.595	.797	.174
V12	*.050	*.002	.198	.126	*.008	*.010
V13	.804	*.002	.856	*.049	.783	.059
V14	*.007	*.010	.724	*.031	.208	*.036
V15	.286	.290	.065	.033	.648	.077
V16	.057	.186	.356	.198	.549	.185

* denotes <.05

Key to mood dimensions on page 211

Key to groups on page 185

Skin Conductance Data

As in the pilot study the skin conductance data were recorded onto magnetic tape and 10 data blocks were isolated by means of the SARA program (Law, 1973). The main variables which were extracted from the data were:

1) onset basal level,
2) offset basal level,
3) response amplitude,
4) rise time,
5) number of responses.

The change scores derived from the difference between the onset basal level and the offset basal level were also computed. All resistance scores were transformed to conductance and the analysis of variance was carried out on the log conductance scores. The results from the analysis are given in Tables 20 to 25, and illustrated in Graphs 5 to 8.

Overall the results indicate that there are no significant group differences, nor are there any significant differences between the conditions of control/non-control or the three levels of instructional set. There is a significant Trials effect (T), $p<.001$, and a significant linear trend across trials (LIN T), $p<.001$, which when graphed shows that there is a tendency towards increased skin conductance across trials. The positive set subjects have a tendency towards a higher skin conductance level overall.

The response amplitude shows that the non-control subjects and the positive set subjects show larger responses than the control subjects, particularly over the first three trials. These results are essentially the same for

the measure of rise time which may well be because these two measures are not necessarily independent. The correlation between rise time and response amplitude is 0.223 $p<.07$. These results do not reach statistical significance however except for a significant trials effect (T), $p<.001$.

There is a significant trials effect for the total number of responses per trial block and a tendency for the negative set subjects to show more responses than the other levels of instructional set.

The measure of skin conductance change shows a significant trials effect (T), $p<.001$, and the non-control condition shows greater change for trials 1 and 8 compared to the control group. The positive set subjects show greater change on trials 1, 6, 7, 8 and 9 compared to other levels of set.

These results show that as far as physiological responding is concerned there is little difference between groups or experimental conditions. It is possible that the information given to the subjects as part of the cognitive set manipulation served to maximise the task importance and brought all groups to the same level. The other possibility is that the experimental manipulations were insufficient to effect differential changes in physiological responsivity. This is unlikely as these manipulations were sufficient to induce behavioural and mood changes. As no pre-instructional measure of skin conductance was taken it is not possible to test the hypothesis that it was the instructions that were responsible for these results. This is unlikely to account for the differences between control and non-control conditions as the instructions were the same. However, it

is possible that the initial differences between the levels of instructional set were due to the instructions. That is, subjects who received the positive set instructions may have been made anxious about how well they would do after being told that the experiment was NOT easy and that most subjects could NOT do it. The anticipation of failure, or rather, the anticipation of the psychological cost of failure is maximised in this condition. So subjects who were positively set at the beginning may have been most anxious and the resultant increased skin conductance may have been maintained throughout the experiment. This is speculative and is not supported by differences in self reported mood as might be expected by such an hypothesis. But there are reasons to question the assumed correspondence between physiological activity and self reported affect which are given in the discussion.

KEY 9. KEY TO SKIN CONDUCTANCE DATA

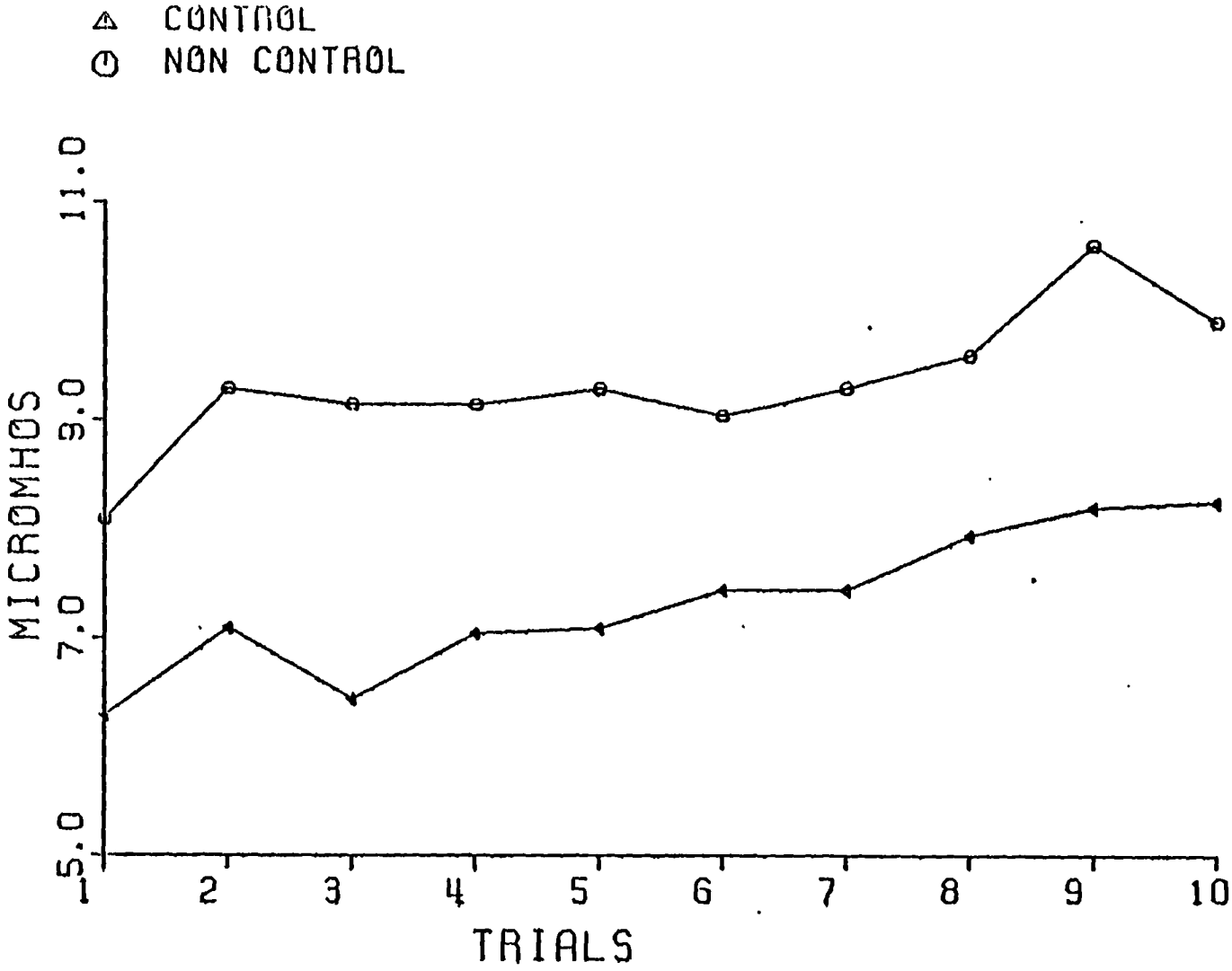
	E1	E2
C1	G3	G6
C2	G2	G5
C3	G1	G4

E1 CONTROL
E2 NON CONTROL
C1 -VE SET
C2 +VE SET
C3 NO SET

TABLE 20. SKIN CONDUCTANCE: ONSET BASAL LEVEL
LOG MICROMHOS

SOURCE	F	d.f.	p
G	0.85	5,54	N.S.
T	18.76	9,486	<.001
G.T	1.12	45,486	N.S.
E	1.61	1,54	N.S.
E.T	0.36	9,486	N.S.
C	0.57	2,54	N.S.
C.T	1.42	18,486	<.05
C.E	0.78	2,54	N.S.
C.E.T	1.19	18,486	N.S.
LIN T	34.07	1,54	<.001
G.W	0.47	5,54	N.S.

GRAPH 5: SKIN CONDUCTANCE ONSET BASAL LEVEL BY CONTROL / NON CONTROL.



GRAPH 6: SKIN CONDUCTANCE ONSET BASAL LEVEL BY LEVEL OF SET.

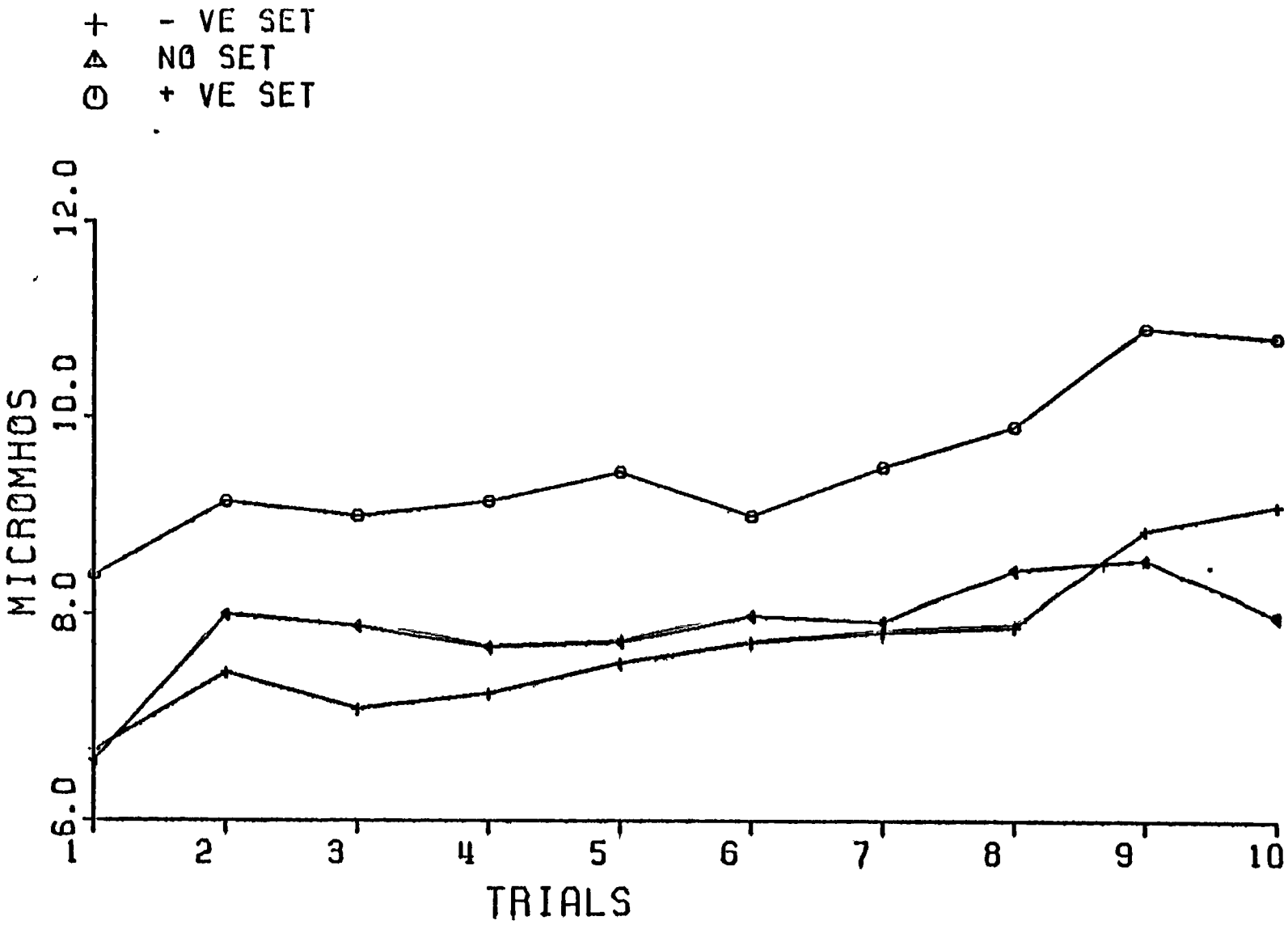


TABLE 21: SKIN CONDUCTANCE: OFFSET BASAL LEVEL
LOG MICROMHOS

SOURCE	F	d.f.	p
G	0.83	5,54	N.S.
T	9.31	9,486	<.001
G.T	1.34	45,486	N.S.
E	1.98	1,54	N.S.
E.T	0.86	9,486	N.S.
C	0.42	2,54	N.S.
C.T	1.55	18,486	N.S.
C.E	0.69	2,54	N.S.
C.E.T	1.38	18,486	N.S.
LIN T	11.36	1,54	<.001
G.W	0.87	5,54	N.S.

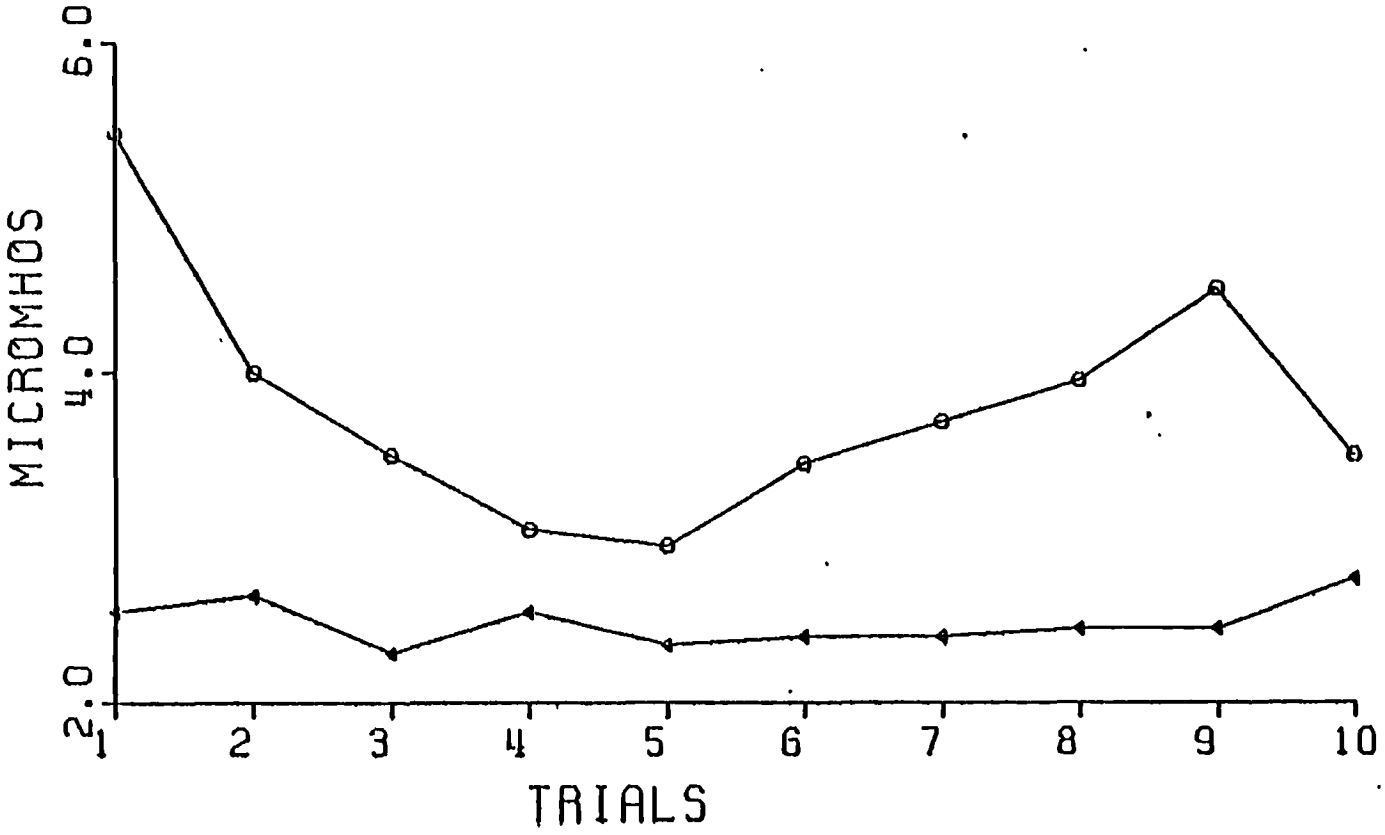
TABLE 22. SKIN CONDUCTANCE: RESPONSE AMPLITUDE
LOG MICROMHOS

SOURCE	F	d.f.	p
G	1.35	5,54	N.S.
T	4.85	9,486	<.001
G.T	0.96	45,486	N.S.
E	2.84	1,54	N.S.
E.T	1.04	9,486	N.S.
C	1.52	2,54	N.S.
C.T	1.15	18,486	N.S.
C.E	0.42	2,54	N.S.
C.E.T	0.72	18,486	N.S.
LIN T	2.01	1,54	N.S.
G.W	1.33	5,54	N.S.

GRAPH 7: SKIN CONDUCTANCE RESPONSE AMPLITUDE BY CONTROL / NON CONTROL.

△ CONTROL

⊙ NON CONTROL



GRAPH 8: SKIN CONDUCTANCE RESPONSE AMPLITUDE BY LEVELS OF SET.

+ NO SET

△ - VE SET

⊙ + VE SET

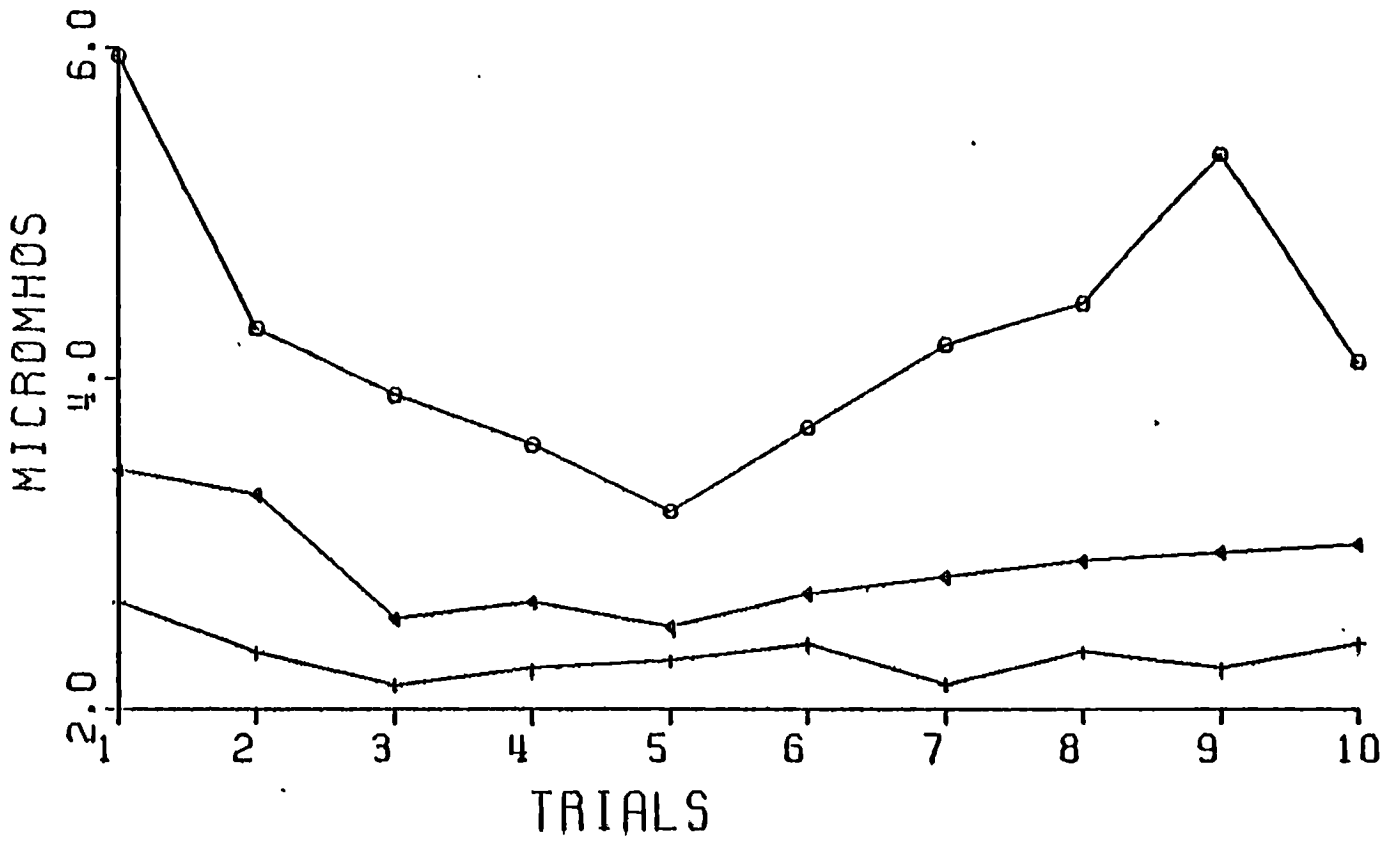


TABLE 23. SKIN CONDUCTANCE: RISE TIME

SECONDS

SOURCE	F	d.f.	p
G	0.68	5,54	N.S.
T	5.04	9,486	<.001
G.T	1.13	45,486	N.S.
E	0.20	1,54	N.S.
E.T	1.44	9,486	N.S.
C	1.36	2,54	N.S.
C.T	1.29	18,486	N.S.
C.E	0.23	2,54	N.S.
C.E.T	0.78	18,486	N.S.
LIN T	2.86	1,54	N.S.
G.W	2.06	5,54	N.S.

TABLE 24. SKIN CONDUCTANCE: TOTAL NUMBER OF RESPONSES (GROUP AVERAGES)

T	E1	E2
1	2.27	2.18
2	2.21	2.42
3	2.59	2.23
4	1.77	1.94
5	1.81	1.78
6	1.58	1.57
7	1.66	1.61
8	1.35	2.33
9	1.41	1.69
10	1.25	1.74

	C1	C2	C3
1	2.55	1.91	2.18
2	2.56	1.78	2.34
3	2.50	2.05	2.17
4	2.06	1.79	1.75
5	1.89	1.66	1.84
6	1.78	1.51	1.44
7	1.72	1.41	1.79
8	2.22	1.77	1.55
9	1.83	1.52	1.31
10	2.28	1.05	1.17

TABLE 25. SKIN CONDUCTANCE: ANALYSIS OF TOTAL NUMBER OF RESPONSES

SOURCE	F	d.f.	p
G	0.57	5,54	N.S.
T	5.08	9,486	<.001
G.T	1.15	45,486	N.S.
E	0.27	1,54	N.S.
E.T	1.52	9,486	N.S.
C	1.04	2,54	N.S.
C.T	0.86	18,486	N.S.
C.E.	0.23	2,54	N.S.
C.E.T	1.34	18,486	N.S.
LIN T	19.20	1,54	<.001
G.W	1.31	5,54	N.S.

Respiration Data

This measure was taken because:-

a) it might provide information concerning the non-significant heart rate data obtained from the first study.

b) it might be of interest as a variable in its own right.

The data were hand scored, taking 60 second trial blocks (30 seconds pre-stimulus and 30 seconds post-stimulus). The time taken for each complete respiratory cycle within this block was measured and a mean pre-stimulus and a mean post-stimulus level was calculated. The difference score between these two measures was also derived and further expressed as a function of the pre-stimulus mean level to serve as a law of initial values correction. The use of a thermistor as a transducer precluded the use of additional measures such as amplitude of the respiratory response as such measures make assumptions about the characteristics of the transducer (e.g. the linearity between the heating and cooling properties of the thermistor and the output into the polygraph).

The results from the analysis reveal a highly significant difference between the pre-stimulus and the post-stimulus scores ($p<.001$) which holds for all groups and all conditions. The pre-stimulus mean level was significantly different between control and non-control (E) with the non-control group having shorter times for the respiratory cycle than the control group, i.e. a higher respiration rate.

All other measures failed to show any significant differences between groups or conditions. The highly significant difference between the pre-stimulus and the post-stimulus measures may be explained as being due to an increase in anxiety or arousal when the trial commences, but it could also be explained as being due to an increase in attention or task involvement during the trial. Similarly, the faster respiration rate for the non-control subjects pre-stimulus may be due to greater anticipatory arousal/anxiety or attentiveness.

The literature on respiration as a psychophysiological measure is not sufficiently adequate to allow further interpretation of the results. The reliable change in respiration rate is of interest and further research may reveal respiration to be a useful index of either arousal or task involvement. The choice between these two alternatives has yet to be established. But, the pilot work carried out prior to this study shows that respiration changes similar to those observed in this experiment are observed when the subject is simply exposed to the noise. Button pushing alone does not cause respiratory change. It seems likely that the highly significant pre-post stimulus changes observed in all groups are due to the noise. To what extent such respiratory change is an index of stress is debatable. Given that stress results from prolonged exposure to the stressor and is to some extent cumulative, then it would be expected that respiratory change across trials would be observed. This does not seem to be the case.

TABLE 26. RESPIRATION: PRESTIMULUS MEAN LEVEL
(Seconds)

T.	E1.	E2.
1	3.94	3.68
2	3.88	3.72
3	3.92	3.43
4	3.94	3.59
5	3.83	3.45
6	3.79	3.51
7	3.85	3.50
8	3.82	3.37
9	4.19	3.49
10	3.87	3.43

	C1.	C2.	C3.
1	4.05	3.71	3.67
2	4.15	3.60	3.65
3	3.93	3.50	3.60
4	4.23	3.43	3.63
5	3.91	3.65	3.38
6	3.86	3.59	3.48
7	3.78	3.55	3.68
8	3.57	3.54	3.67
9	4.08	3.65	3.78
10	3.69	3.50	3.76

TABLE 27. RESPIRATION: ANALYSIS OF PRESTIMULYS MEAN LEVEL

SOURCE.	F.	d.f.	p.
G	1.42	5,46	N.S.
T	1.27	9,414	N.S.
G.T	1.00	45,414	N.S.
E	4.13	1,46	<.05
E.T	0.94	9,414	N.S.
C	1.31	2,46	N.S.
C.T	1.30	18,414	N.S.
C.E	0.44	2,46	N.S.
C.E.T	0.73	18,414	N.S.
LIN T	0.54	1,46	N.S.

TABLE 28. RESPIRATION: POST-STIMULUS MEAN LEVEL (seconds)

T	E1.	E2.
1	2.25	2.26
2	2.17	2.08
3	2.21	2.12
4	2.27	2.17
5	2.21	2.12
6	2.21	2.17
7	2.79	2.23
8	2.23	2.21
9	2.32	2.19
10	2.89	2.19

	C1.	C2.	C3.
1	2.15	2,32	2,30
2	2.11	2.16	2.12
3	2.17	2.18	2.16
4	2.27	2.19	2.20
5	2.04	2.24	2.21
6	2.14	2.21	2.21
7	2.18	2.35	2.34
8	2.06	2.24	2.35
9	2.14	2.33	2.30
10	2.17	2.92	2.35

TABLE 29. RESPIRATION: ANALYSIS OF POST-STIMULUS MEAN LEVEL

SOURCE	F.	d.f.	p.
G	1.21	5,46	N.S.
T	2.05	9,414	N.S.
G.T	1.01	45,414	N.S.
E	0.45	1.46	N.S.
E.T	0.48	9,414	N.S.
C	0.32	2,46	N.S.
C.T	0.95	18,414	N.S.
C.E	2.42	2,46	N.S.
C.E.T	1.38	18,414	N.S.
LIN T	2.30	1,46	N.S.

TABLE 30. RESPIRATION CHANGE (PRE - POST) SECONDS

T.	E1.	E2.
1	1.69	1.42
2	1.70	1.64
3	1.71	1.30
4	1.67	1.40
5	1.63	1.33
6	1.57	1.34
7	1.57	1.27
8	1.58	1.16
9	1.88	1.29
10	1.53	1.24

	C1.	C2.	C3.
1	1.91	1.40	1.45
2	2.05	1.45	1.54
3	1.76	1.32	1.45
4	1.95	1.25	1.44
5	1.86	1.41	1.17
6	1.71	1.39	1.27
7	1.60	1.20	1.45
8	1.51	1.30	1.32
9	1.94	1.33	1.48
10	1.53	1.21	1.42

TABLE 31. ANALYSIS OF RESPIRATION CHANGE (PRE - POST) SECONDS

SOURCE	F.	d.f.	p.
G	1.52	5,46	N.S.
T	1.32	9,414	N.S.
G.T.	0.85	45,414	N.S.
E	3.16	1,46	N.S.
E.T	0.69	9,414	N.S.
C	2.48	2,46	N.S.
C.T	0.76	18,414	N.S.
C.E	0.05	2,46	N.S.
C.E.T	1.02	18,414	N.S.
LIN T	2.72	1,46	N.S.

ANALYSIS OF PRE - POST

E = 49.62 18d.f. $p<.001$

TABLE 32. RESPIRATION CHANGE AS A FUNCTION OF PRESTIMULUS LEVEL

T.	E1.	E2.
1	0.070	0.058
2	0.070	0.069
3	0.085	0.060
4	0.067	0.062
5	0.067	0.061
6	0.068	0.060
7	0.064	0.057
8	0.066	0.056
9	0.071	0.058
10	0.063	0.056

	C1.	C2.	C3.
1	0.074	0.061	0.058
2	0.076	0.066	0.067
3	0.070	0.061	0.060
4	0.074	0.058	0.062
5	0.076	0.061	0.054
6	0.071	0.063	0.056
7	0.068	0.054	0.061
8	0.067	0.061	0.056
9	0.073	0.060	0.061
10	0.066	0.055	0.058

TABLE 33. ANALYSIS OF RESPIRATION CHANGE AS A FUNCTION OF PRESTIMULUS LEVEL

SOURCE	F.	d.f.	p.
G	1.50	5,46	N.S.
T	1.47	9,414	N.S.
G.T	0.85	45,414	N.S.
E	2.19	1,46	N.S.
E.T	0.67	9,414	N.S.
C	2.11	2,46	N.S.
C.T	0.55	18,414	N.S.
C.E	0.80	2,46	N.S.
C.E.T	1.24	18,414	N.S.
LIN T	4.51	1,46	<.05

Heart Rate Data

This variable was not analysed in any detail. There were problems concerning interference from 50 Hz mains sources which were corrected. The heart rate recordings obtained contained a proportion of missing trials due to muscle interference. The major problem was concerned with the nature of the noise stimulus and the task the subject was required to perform in order to attempt to control the noise. The impact of the noise caused a 'flinch' reflex which rendered the first few seconds of the record after the noise onset unscorable. This effect was probably maximised by the requirement for the subject to start pressing the button as quickly as possible after the onset of the noise. A variety of electrode placements were tried but this effect persisted. The net result was that a large proportion of the heart rate data was unscorable. A hand scoring technique was used to measure individual inter beat intervals from the chart records for a sample of the subjects and a similar pattern to that of the first study emerged. That is, where responses to trial onset did occur, i.e. cardiac acceleration or deceleration, these responses were not reliable in their direction. For some trials an acceleration was observed, for some trials a deceleration. In many cases there was no observable response. When the data were pooled across trials or across subjects the heart rate responses tended to cancel each other out.

In view of the detailed analyses employed in the first study and the failure to identify a heart rate measure or analysis which discriminated between the experimental conditions, the value of measuring heart rate as a dependent

variable is dubious. As mentioned before, it is possible that the nature of the learned helplessness experiment precludes the use of such a sensitive measure as heart rate. Given the tight homeostatic control of cardiac activity and the fact that subjects are required to tolerate relatively high noise levels as well as effecting a rapid motor response, perhaps it is unreasonable to assume that a consistent differentiation between experimental conditions will emerge. This point will be brought up again in the overview of the physiological data.

Miscellaneous Data

The miscellaneous data includes the information gathered about the subject sample from personality questionnaires etc. and also various items such as ratings of the aversiveness of the stimuli employed in the experiments, motivation etc. The first major datum to be considered is the attribution self reported by the subject at the end of the experiment regarding their performance on the pre-treatment noise task. Table 34 gives these results and it can be seen that subjects characteristically form specific, internal, and stable attributions concerning their performance, i.e. that their performance was due to their ability on this particular test. Subjects in G1 (-ve set control), however, tended to attribute their performance to a specific, external, stable factor. That is, their performance was due to their increased motivation contingent upon their perception that the tests were particularly relevant and not to their ability per se.

There are two major implications from these data. Firstly, that the information and feedback in general did not significantly affect subjects' attributions as much as they affected the perception of the success/failure dimension previously defined. The data from G1 is a notable exception. This supports the assumption that the perception of success/failure is not just simply a function of forming the correct attributions. Secondly, the subjects' attributions were fairly homogeneous, which brings into question the necessity of an attributional component in human experiments. EXCEPT FOR THE FACT THAT it would be predicted that it would be the subjects who formed the internal attributions that would become the most helpless, and the subjects in this experiment tended to form internal attributions and also highly significant helplessness effects were found. It should be noted that the attributional questions that the subjects were required to check are difficult to formulate in the first place and it cannot be guaranteed that all these questions are equivalent on the basis of their relevance, appropriateness, etc. It does seem that the set and feedback manipulations served to maximise task importance, affected the subjective self report of the perception of success/failure without affecting the attributions of actual performance to a significant degree, and as such provide good support for the effectiveness of the underlying dimension of uncontrollability in determining the behavioural deficits.

The Mill Hill I.Q. test scores show that the H1 condition (-ve set) had a higher I.Q. than the other levels of H. The P2 (non-control) condition had a higher mean

Rotter Locus Of Control, i.e. were more external, than the P1 condition (control). The H2 (+ve set) condition had a higher P score on the Eysenck Personality Questionnaire than the other levels of H. The non-significant correlations between these scores and such measures as the anagram scores do not support the hypothesis that these sample characteristics significantly affected the overall results. The remainder of the data do not reach significance and the groups are fairly homogeneous on the ratings etc. However, it is worth noting that subjects in the H1 and H2 conditions (-ve and +ve set conditions) were more highly motivated to do the pretreatment than the H3 (no set condition), supporting the hypothesis that these levels of set maximised task importance.

TABLE 34. ATTRIBUTION OF PERFORMANCE

	A	B	C	D	E	F	G	H
G1	0	1	1	1	1	7	0	1
G2	11	0	0	0	0	1	0	0
G3	6	0	0	0	2	2	0	1
G4	10	0	1	0	0	0	0	1
G5	3	0	1	2	3	1	0	1
G6	11	1	0	0	0	0	0	0
P1	9	1	2	3	6	10	0	3
P2	32	1	1	0	0	1	0	1
H1	11	1	1	1	1	8	0	1
H2	16	0	1	0	2	2	0	2
H3	14	1	1	2	3	1	0	1

A)	SPECIFIC	INTERNAL	STABLE
B)	GLOBAL	INTERNAL	STABLE
C)	GLOBAL	INTERNAL	UNSTABLE
D)	SPECIFIC	INTERNAL	UNSTABLE
E)	GLOBAL	EXTERNAL	STABLE
F)	SPECIFIC	EXTERNAL	STABLE
G)	GLOBAL	EXTERNAL	UNSTABLE
H)	SPECIFIC	EXTERNAL	UNSTABLE

N.B. The exact questions are given in the appendix.
See Post Experimental Questionnaire for this study.

KEY 10: KEY TO MISCELLANEOUS DATA

AGE	:	YEARS
BECK	:	DEPRESSION SCORE
ROTTER	:	LOCUS OF CONTROL SCORE
MHIQ	:	MILL HILL I.Q. SCORE
P	:	PSYCHOTICISM
E	:	EXTRAVERSION/INTROVERSION
N	:	NEUROTICISM/STABILITY
L	:	LIE SCALE
NRP	:	NOISE RATING ON PLEASANTNESS DIMENSION *
NRL	:	NOISE RATING ON LOUDNESS DIMENSION *
DETN	:	DETERMINATION ON PRETREATMENT *
DETA	:	DETERMINATION ON TEST TASK *
AEXP	:	ANAGRAM SOLVING EXPERIENCE *

*High scores indicate more unpleasant
louder
lower determination
less experience

TABLE 35: MISCELLANEOUS DATA

		AGE	BECK	ROTTER	MHIQ	
G1	MEAN	21.80	3.83	12.17	119.89	
	S.D.	3.22	4.69	4.75	8.62	
G2	MEAN	22.78	3.09	14.44	118.78	
	S.D.	4.44	4.59	3.36	6.70	
G3	MEAN	24.30	5.10	10.08	114.00	
	S.D.	5.30	4.91	4.42	10.70	
G4	MEAN	22.00	3.75	13.54	114.13	
	S.D.	4.54	2.93	4.16	11.85	
G5	MEAN	21.64	5.33	12.25	110.82	
	S.D.	1.86	6.32	3.93	10.97	
G6	MEAN	21.92	6.25	13.25	109.18	
	S.D.	4.55	6.81	3.08	7.29	
H1	MEAN	22.26	3.47	13.14	119.33	
	S.D.	3.76	4.55	4.27	7.51	
H2	MEAN	23.28	4.36	11.74	114.06	$p<.01$
	S.D.	5.03	3.91	4.55	10.88	
H3	MEAN	21.77	5.79	12.75	110.00	
	S.D.	3.39	6.44	3.49	9.10	
P1	MEAN	22.54	4.74	11.50	113.60	
	S.D.	3.81	5.25	4.37	9.29	
						$p<.05$
P2	MEAN	22.21	4.40	13.69	114.16	
	S.D.	4.35	5.09	3.48	10.55	

TABLE 35 (contd.)

EPQ		P		E	N	L
G1	MEAN	2.86		13.50	12.25	5.50
	S.D.	1.73		6.46	4.37	4.41
G2	MEAN	1.83		12.83	10.50	4.50
	S.D.	1.94		6.27	6.38	2.74
G3	MEAN	4.00		13.00	13.22	5.22
	S.D.	2.78		4.06	5.99	3.87
G4	MEAN	3.40		13.80	10.00	3.80
	S.D.	3.21		4.55	2.45	2.49
G5	MEAN	2.10		14.50	11.10	4.60
	S.D.	1.60		4.45	8.03	3.20
G6	MEAN	2.44		13.00	11.89	6.11
	S.D.	1.33		3.74	4.57	3.33
H1	MEAN	2.43		13.21	11.50	5.07
	S.D.	1.82	p<.01	6.14	5.17	3.69
H2	MEAN	3.78		13.29	12.07	4.71
	S.D.	2.83		4.08	5.15	3.41
H3	MEAN	2.26		13.79	11.47	5.32
	S.D.	1.45		4.09	6.46	3.27
P1	MEAN	2.96		13.70	12.15	5.07
	S.D.	2.17		4.86	6.27	3.68
P2	MEAN	2.50		13.15	11.00	5.05
	S.D.	2.06		4.56	4.63	2.99

TABLE 35 (contd.)

		NRP	NRL	DETN	DETA
G1	MEAN	73.75	66.66	11.00	16.65
	S.D.	17.08	15.23	11.38	18.77
G2	MEAN	67.00	62.50	13.67	12.67
	S.D.	13.76	11.22	13.75	15.72
G3	MEAN	75.50	70.67	7.92	16.33
	S.D.	18.87	16.90	5.57	11.80
G4	MEAN	64.00	61.25	18.75	17.08
	S.D.	18.14	16.12	14.68	11.05
G5	MEAN	68.83	67.83	22.33	16.83
	S.D.	12.65	14.92	27.29	16.08
G6	MEAN	74.16	69.67	21.00	15.92
	S.D.	24.89	19.28	22.80	18.81
H1	MEAN	70.37	65.08	12.33	14.71
	S.D.	15.56	13.47	12.42	17.01
H2	MEAN	69.75	65.95	13.33	16.71
	S.D.	19.03	16.85	12.19	11.18
H3	MEAN	71.50	68.75	21.67	16.38
	S.D.	19.50	16.89	24.61	17.07
P1	MEAN	72.69	68.72	13.75	16.64
	S.D.	16.19	15.32	18.00	15.32
P2	MEAN	68.38	64.47	17.81	15.22
	S.D.	19.40	15.58	17.23	15.15

TABLE 35 (contd.)

		AEXP
G1	MEAN	57.92
	S.D.	31.98
G2	MEAN	80.17
	S.D.	16.18
G3	MEAN	72.42
	S.D.	20.46
G4	MEAN	71.58
	S.D.	25.13
G5	MEAN	55.75
	S.D.	29.62
G6	MEAN	69.17
	S.D.	30.88
H1	MEAN	69.04
	S.D.	27.27
H2	MEAN	72.00
	S.D.	22.42
H3	MEAN	62.46
	S.D.	30.38
P1	MEAN	67.03
	S.D.	28.03
P2	MEAN	73.63
	S.D.	24.56

DISCUSSION

Discussion

The purpose of the main study was to test the theory of learned helplessness against the alternative hypothesis, which was that the deficits observed are due to interference resulting from the perception of failure. Interference refers to feelings of dysphoria, discontent, and thoughts which distract the subject and prevent the subject from performing optimally on the test task. This is distinctly different from the central learning deficit which is an important component of the theory of learned helplessness. The reformulation of the theory subsumes failure as a concept under helplessness which occurs within certain specified attributional circumstances. The validity of this is questionnable as there is evidence and opinion (Coyne et al., 1980) that suggest that failure may be distinctly different from the learned helplessness mechanism. It is also of relevance that the design and partial execution of this study was effected prior to the publication of the reformulation and also that the failure/uncontrollability controversy is still a current feature of the literature.

The reformulation incorporates failure to the extent that it is necessary to withdraw from helplessness theory altogether in order to view failure as a separate concept. Failure refers here to a subjective evaluation and is implemented as an experimental variable by operational definition. In this study, control and non-control were effected by information given to the subject which led the subject to believe that his behaviour and the experimental outcome were veridical. In fact they were not. This manipulation has been used before in the literature and also

in this experiment quite successfully. Similarly, success and failure were effected by information given to the subject concerning how the subject was to evaluate his performance relative to a supposed population. In the first case, i.e. control, the information tells the subject whether he had control or not over the stimulus. In the second case, i.e. failure, the information tells the subject whether he succeeded or failed the test. The design of the study included a no set condition which acted as a control for conditions of control and non-control without manipulation of success/ failure evaluation. It is possible to assess the effects of instructional set by comparing the cell in question with the appropriate no set cell whether it is control or non-control.

The predictions from this experiment are that if non-control determines deficits and the instructional set is irrelevant, then significant main effects for non-control should be apparent. Contrariwise, if significant effects are found for instructional set and not for non-control then instructional set is the more important factor. Significant interaction terms would indicate that both instructional set and non-control are important and both can affect the performance of the subject.

The results from this study are fairly clear in so far as the data from the anagram test task are concerned. There are highly significant differences between the experimental conditions of control and non-control, with the non-control groups showing the poorest performance. There were no significant differences between the various levels of instructional set. The self report data on this manipulation

indicate that subject in the positive set and negative set conditions tended to report success or failure as being more important than control or non-control. The frequency of this self report does not differ significantly between conditions. A breakdown analysis which compared subjects reporting control/non-control with subjects reporting success/failure failed to reveal any significant differences between these groups. It is noted that, in general, the negative set non-control group subjects did worse than other groups of subjects but this is not reflected in any significant interaction terms.

These results are strongly supportive to the theory of learned helplessness in that the major determinant of the deficits is non-control and not the alternative condition of instructional set. It is possible to explain this perception of success or failure in terms of the reformulation. That is, the levels of instructional set affect the attributions that are made by the subject regarding their perception of control or non-control. Table 34 gives the self report data concerning the subjects attributions about their performance on the pretreatment phase of the experiment. It seems that subjects in general form an internal, specific, and stable attribution. That is, they attribute their performance to their own inability on this particular task. Subjects in group 1 (negative set control) tend to report a specific, external, and stable attribution, i.e. their performance was a function of the relevance and fairness of the test. There seems to be no clear cut relationship between the levels of instructional set and the attributions formed by the subjects. It seems likely that when such factors as task importance and relevance are maximised so that the subjects are highly

motivated and have an adequate context within which to interpret their performance, the attributional components are largely redundant as they tend to be held constant within the confines of the experiment.

It is factors such as these that may have contributed to the skin conductance results which reveal no significant differences between groups or between conditions, save for a tendency for skin conductance level to increase over the experiment against the 'normal' expected habituation trend.

The mood affect check list data are strongly supportive to the learned helplessness theory in that there are significant differences between control and non-control conditions, with the non-control groups showing 'negative' mood changes after the pretreatment phase of the experiment which remit after the test task. The significant differences between the relevant groups show the effectiveness of the instructional set manipulation and also show that the mood changes were more profound for the HI negative set subjects.

The respiration data show a highly significant difference between the prestimulus measure and the poststimulus measure but no difference otherwise except that the P2 condition (non-control) show a significantly higher respiration pre-stimulus. The pre/post stimulus differences may be regarded as being due to an increase in arousal/anxiety during the trials. It is also likely that the prestimulus condition differences are due to a similar increase in arousal. There are other plausible alternatives however, such as increased attention, task involvement, etc..

The heart rate data were heavily contaminated by movement artifacts and flinch reflexes due to the onset of the noise. Those subjects records which could be analysed failed to show any consistent pattern. It was impossible to analyse these data systematically on their own let alone to take respiration rate as a covariate of heart rate.

Several points arise from the miscellaneous data. Firstly, the P2 group (non-control), were more external on the Rotter Locus Of Control Questionnaire, but the overall correlation with their performance on the anagram test task was statistically non-significant and so it is not possible that this personality dimension can account for the group differences on the anagram solving task. Subjects in the negative set condition scored more highly on the Mill Hill I.Q. test but again the correlation between I.Q. and anagram solving performance did not reach statistical significance. The positive set subjects had significantly higher P scores on the Eysenck Personality Questionnaire. Finally, the subjects self reported experience with solving anagrams correlated highly with anagram solving ability on the test task. This was not used as the basis of a further breakdown analysis or as a data weighting as this particular datum was obtained after the anagram solving test task had been completed by the subject and is probably highly contaminated by the subjects evaluation of her own performance.

To summarise, the results from this study clearly demonstrate deficits associated with non-control as would be predicted by the original theory of learned helplessness. The alternative explanation in terms of the perception of failure on a psychology test, as defined by the manipulation of instructional set, is unsupported. The self report data indicate that the levels of instructional set were effective manipulations, but they were not a significant determinant of performance deficits nor did they outweigh the effects of the dimension of uncontrollability.

CHAPTER 8

DISCUSSION

It is the purpose of this chapter to state succinctly the findings from the experimental studies and to return to the literature review and to establish what bearing these results have on the theory of learned helplessness.

The first study investigated a standard helplessness triadic design but used non-veridical feedback to induce the perception of control or non-control with a heterogeneous subject sample. Deficits were observed on an anagram test task in the groups that had been exposed to uncontrollable aversive noise. In general, these results failed to reach statistical significance. The skin conductance data and the mood affect check list data indicated that the groups exposed to the uncontrollable contingency became more aroused physiologically as the experiment progressed (compared to the other groups which showed habituation trends), and self reported more negative mood change. The poor performance of the passive control groups on the anagram test task was explained as being due to lack of task involvement in the pretreatment phase and a function of rating the noise as more unpleasant compared to the other groups. The results were considered to be supportive to the theory of learned helplessness, and it was argued that the deficits were attenuated by the heterogeneity of the subject sample.

The second study was larger and was designed to test the alternative explanation of the human data that the deficits on the test task were due to dysphoria, anxiety, and lack of motivation caused by the perception of failure as opposed to the perception of response outcome independence. The perception of failure/success was manipulated by the use of different levels of instructional

set and trial by trial feedback. This experiment involved a pretreatment using non-veridical perception of control/non-control over loud noise crossed with three levels of instructional set. The anagram results showed highly significant differences between the control/non-control conditions supportive to the theory of learned helplessness and no significant differences between the levels of set. The self report data and the poor performance of the negative set, non-control group indicated that the manipulations were effective i.e. affected the subjects' perception of the experiment, and so the results refute the success/failure alternative. The mood affect check list data were also supportive to this, in that non-control subjects showed the greatest negative mood changes, this being most evident in the negative set non-control group. The physiological data failed to discriminate between groups or conditions and indicated that all subjects maintained a high level of arousal throughout the experiment. For example, each group showed a gradual increase in skin conductance level as the experiment progressed. The respiratory rate increased significantly at the onset of the noise for each trial with no significant habituation of this response across trials or between groups or conditions.

These results are strongly supportive to the theory of learned helplessness but also have important implications concerning the cognitive level of explanation and the reformulation which incorporates attributional components. Whilst the levels of set affected subjects' perception of failure or non-control, the attributions concerning the

reason for their performance remained constant. This could be due to the fact that task importance was maximised in the experiment and that subjects were led to believe that their performance was a function of their I.Q. But, this could also be the case for other laboratory experiments using human subjects and would cast doubt on the appropriateness of the social learning reformulation as far as these experiments are concerned. When the implications of helplessness theory to everyday life and clinical depression are considered then the attributional components are clearly important. An uncontrollable outcome will only have effect given that the outcome is important and that an appropriate attributional context is formed. But, there are several problems with the attributional components that have been identified by Wortman & Dintzner (1978). These are as follows:-

1) firstly, do people make attributions and are they significant determinants of behaviour ?

2) are the attributional dimensions selected for the reformulation the correct ones ?

Given that attributions are important and that the relevant dimensions have been selected, there is still no provision in the reformulation for the meaning of the outcome and no account for the predictability factor. That is, the importance of the outcome is a critical determinant of the extent to which helplessness deficits will be observed. A trivial outcome, for example, control over a tone or light stimulus, is unlikely to produce debilitation unless it has some meaning or importance to the person, such as in the context of a psychology experiment or as a

signal for a malfunction in a machine. In both cases, the actual stimulus, which is essentially trivial, acquires a significance due to the context in which it occurs. The attributional reformulation should be able to provide predictions about situational factors which affect the significance of the event as well as the causal attribution for the overall control of the event. It is also necessary to state explicitly the conditions under which attributions are formed otherwise the model becomes circular.

Referring back to the basic issues stated in Chapter 4, i.e. the discrepancies between the animal and the human experiments, the necessity of postulating cognitive representation of contingencies etc., it is possible to identify various levels of data and correspondingly, several levels of theory structure:-

A) ANIMAL EXPERIMENTS:- these experiments have been tightly designed along the lines of classical operant conditioning studies and have yielded consistent results. The alternative explanations to the theory of learned helplessness have tended to be S-R interpretations and whilst these alternatives are inadequate, a more formal S-R version of the theory of learned helplessness would be sufficient to account for the data (as in Maier & Seligman, 1976). The problems of definition and the tendency to anthropomorphise outweigh any advantage that a cognitive learning model may have to offer.

B) HUMAN EXPERIMENTS:- these experiments have developed along the lines of the animal research and the same argument applies with the proviso that, given the increased behavioural repertoire of the organism,

some account of individual differences must be made. The necessity of postulating and validating a cognitive model with attributional components is a debatable and important issue at this level of data. To expand upon this point, self report data provide support that contingencies can be verbalised and that cognitive structuring of the event can occur. Whilst it is desirable to allow for these data in the theory, it is essential to consider the extent to which the original theory, which places major emphasis on the environmental determinants of the deficits is sufficient. Further, to consider the extent to which inclusion of cognitive factors detracts from the scientific necessity for testable hypotheses. This level of data is a critical transition from animal learning experiments to clinical and human learning experiments. The inclusion of cognitive factors must be effected critically and with empirical validation wherever possible.

C) EXTRAPOLATION:- this refers to the clinical data on depressives and the more or less anecdotal evidence cited by Seligman (1975), relating to real life experience. Here the situation is not structured and controlled as in the laboratory experiments but is an application of the basic model. In order for this application to be practical, it is necessary to define and categorise situations where helplessness is an appropriate model. The cognitive model with relevant attributions is more or less essential to define subjectively those circumstances under which uncontrollability can result in behavioural deficits.

It can be seen that as the level of data becomes less tightly defined by the experimenter, it is necessary to modify the level of theorising to account for the increased variability of the basic data. The main issue to be considered relates to the intermediate level i.e. the human experiments. The data collected from the second study is directly relevant to the issue of whether a cognitive learning model is necessary and the extent to which an attributional superstructure is in evidence.

The second study was designed to test an alternative explanation to the perception of non-control, i.e. that the deficits are due to the perception of failure by the subject. It is worth while differentiating between 'the perception of failure' by the subject and 'the perception of non-control' by the subject. The theory of learned helplessness states that deficits will be observed in subjects exposed to uncontrollable stimulation given that a perception of response outcome independence is formed. The extent to which the deficits are expressed behaviourally is in part a function of the attributions of causality for the lack of control. This assumes a continuity between the animal and the human experiments. The alternative 'perception of failure' hypothesis suggests that there is a disparity between the animal and the human experiments. That is, the human experiments involve the perception of failure (i.e. the subject perceives that he cannot effect control but believes that others can effect control). It was suggested that this perception of failure was instrumental in determining the deficits rather than the perception of non-control. This differs from the

reformulation account of personal helplessness in that it does not assume the human experiments to be a natural extension of the animal experiments, but proposes a different unique cause for the deficits. It could be argued that learned helplessness proposes a maladaptive learned state for the deficits, whilst the failure hypothesis proposes that human deficits are due to negative self appraisal. Results supportive to the failure hypothesis would undermine the validity of learned helplessness theory in the human experiments as the underlying mechanism would be different and the role of uncontrollability would be minimal in comparison to the role of situational factors affecting self appraisal. The results suggest that the perception of non-control is the most salient aspect of the experiment to the subject. The self report data indicated that subjects tended to form specific, internal, stable attributions concerning their performance, i.e. their performance was due to their ability on this particular task. This consistency reflects that either subjects formed this particular attribution or that the other alternatives available to the subjects were regarded as being inappropriate. Given the specificity of the experimental situation it is difficult to formulate appropriate attributional choices that cover all possible combinations. Subjects tend to have difficulty in reporting attributions if they are not provided with a list of alternatives. It is possible that the alternatives were inadequate, but it is more likely that this specificity of the subjects responding was due to the specificity of the experimental situation, especially as subjects were given detailed information regarding the purpose of the experiment.

The negative and positive levels of instructional set did have behavioural consequences over and above the effects of control and non-control. Comparison of these groups with the no set groups revealed that positive set served to reduce the negative affective changes brought about by uncontrollability whilst negative set served to increase these negative affective changes. However, whilst these changes are evident they do not contribute significantly to the learning deficits on the anagram test task. It seems that the learning deficits are determined by the uncontrollability of the outcome and the affective components may be seen as behaviours which parallel the deficits. The affective changes are caused by both controllability and instructional set although there are qualitative differences between the exact affective change effected. Referring back to page 75(b) these results suggest that there is a fairly direct pathway between uncontrollability and learning deficits and further that the mood changes observed are parallel behaviours and are not necessarily causal determinants of the learning deficits. This is further supported as the negative mood changes brought about by negative set which were highly significant did not result in significant deficits on the learning task. This is an interesting point as it raises the issue of whether subjects are aware of the non contingency and it is this awareness that produces the deficits.

There are three main problems which arise if this issue is to be considered, and these will remain unresolved.

Firstly, what is meant by the term awareness? What measures of awareness can be taken? Can it be shown that

awareness significantly affects the results over and above the stimulus contingencies defined by the experimenter?

Awareness may be regarded as an inferred construct meaning that environmental stimuli are registered cognitively, but further, that these contingencies are available to the subject for self report. If a subject reports, "I was unable to control the situation", then one may assume that the subject is aware of the stimulus contingencies to which he was exposed. If a subject is unable to make such a self report, it could be that the subject is unaware but the stimulus contingencies could still be registered cognitively. Whilst it can be argued that it is essential for cognitive representation of the contingencies to occur for deficits to be produced, it remains unclear whether the awareness of the subject is a necessary condition for such learning deficits to occur. It is possible that subjects do not wish to report this information as they may regard it as being self evident or, by a process of cognitive dissonance they might deny that they were unable to exert control.

Given that subjects do report the appropriate awareness of the relevant contingencies, the role of this awareness as a determinate of the deficits remains uncertain. It is perhaps, similar to the affective changes associated with uncontrollability where awareness may not cause the deficits but is an epiphenomenon or it may be that this awareness disrupts the subjects performance on the test task by acting as a distraction so that the subject is not devoting all his attention to the test task as he otherwise would. The evidence from the second study shows that exposure to uncontrollability causes deficits on the anagram test task

and associated mood changes. The instructional set did produce mood changes relative to the no set condition but was not a significant determinant of test task deficits.

Whilst it can be argued that behaviour change due to uncontrollability is mediated by cognitive representation by definition (i.e. if it was not represented cognitively then the contingencies would not be affecting the subject), it is unclear the extent to which awareness is a necessary condition. It is also unclear whether awareness is a causal link or a parallel behaviour to the deficits and it is also unclear exactly what the role of attributions are for precisely the same reason. The self report data from the second study show that subjects were able to respond to a forced compliance check list regarding their own awareness of non-control or failure and presumably were aware of the fact that they were unable to do the task. The forced compliance check list for attribution of causality produced fairly consistent results across subjects, i.e. that the subjects performance was due to their ability on the task. Whilst there are studies reporting behavioural consequences from attributions it is unclear what role attributions have in this case. This may reflect the inadequacy of the check list or the fact that attributions are not such as important factors as has been previously suggested. It has been noted previously in this thesis that the attributional dimensions relate to the chronicity, specificity, and other characteristics of helplessness and there are many experimental questions to be answered before the adequacy of the attributional reformulation can be assessed. This thesis does not deal with the attributional reformulation or its predictions directly and so references to the reformulation are tentative.

To summarise the main points of the argument so far:-

1) the two experiments have yielded results strongly supportive to the theory of learned helplessness;

2) the alternative explanation in terms of deficits due to the perception of failure induced by instructional set has not been supported;

3) three levels of theorising have been identified in the learned helplessness model;

4) the level of explanation necessary to explain the data is a function of the degree to which constraints on the situation are systematically applied by the experimenter.

Before concluding, several additional points need to be made. Firstly, to what extent are the physiological variables informative regarding the efficacy of the experimental manipulations ? How do such measures relate to other dependent variables such as self report data ? Can the interaction between these variables provide any information about the level of theory that would be appropriate for any given data ?

The skin conductance data in the first study showed a fairly clear discrimination between groups and corresponded well with the self report data. This was not the case in the second study where this measure failed to discriminate between groups. The self report data did differentiate groups. The essential difference between these two studies was the extent to which they were structured. The information given to the subjects in the second study was far more detailed than in the first study and it was argued that this, coupled with maximising the task importance, served to diminish physiological differences. Another factor which may be informative on the issue of the non correspondence between physiological variables and self report data lies in the discriminability of mood states. A self reported state of pleasure may be congruent with a physiological reaction of fear, an example of which is a pleasurable pastime which incorporates a dimension of danger e.g. hang gliding. Thus mood states may not be independent. The main point is that physiological variables and self report variables both interact and reflect essential differences in the level of responding. In an experiment where tight control is maintained over the physical parameters of the stimuli and

the subject is not required to structure or interpret the situation, physiological responses are of prime importance. The self report data serve mainly as a manipulation check. In the learned helplessness studies described in this thesis, the essential experiment is more complex and the subject is more than a passive transducer between the electrodes and the stimuli. The subject is actively task involved, evaluating his performance, and coping with non control. Another important factor is that both these studies used non-veridical feedback as an experimental condition, so that a major factor is the interpretation of the feedback by the subject, not just the registration of a light stimulus on a sensory receptor.

These points lead to two considerations. First of all, of what value are physiological data in learned helplessness experiments ? Based on the studies in this thesis, it seems that the learned helplessness experiments involve such a high degree of active involvement by the subject, both physically and cognitively that physiological measures are of limited value. The experiment maximises measurement problems, the theory which incorporates concepts of anxiety and depression, makes any group differences obtained difficult to interpret. This leads to the second consideration, what level of explanation is necessary to explain the human experimental data ? The original cognitive model of the perception of response outcome independence. The reasons for this are:-

1) studies using non-veridical feedback have shown that experimental results are obtained consistent with theoretical predictions. That is, it is the subjects

perception of the experiment that is important not the physical parameters of the impinging stimuli;

2) the self report data show significant discrimination between groups where physiological variables fail to discriminate. Disregarding problems of physiological measurement, this implies that systems higher than basic physiological systems are significantly involved i.e. mood states (this does not imply that mood states are completely independent of physiology);

3) the experimental complexity. A 'strictly formal' conditioning model, e.g. classical conditioning, is inadequate as the transference to other tasks requires constructs of anticipation, expectation, which are cognitive.

The simplest adequate theory to explain the human experimental data is the original perception of response outcome independence model.

Therefore, it is suggested that given adequate control over the parameters of the experiment i.e. the task importance and a meaningful context within which the test tasks are presented, an explanation of the deficits in terms of environmental contingencies with cognitive representation is sufficient to explain the basic deficits. The attributional components in the reformulation need to be examined carefully and tested by means of controlled experimentation. The evidence so far is too dependent on the post hoc interpretation of experients rather than testing predictions from the reformulation.

In conclusion, the experimental studies have yielded two main points. Firstly, the theory of learned helplessness has been strongly supported, whilst the alternative explanation in terms of the perception of failure induced by instructional set has not been supported. The evidence suggests that the manipulation of instructional set was effective in the induction of failure perception and certainly affected subjective feelings self reported by the subjects, but was not a significant determinant of learning deficits.

Secondly, the results indicate that subjects in this experiment showed similar attributions concerning the cause of the uncontrollability. Whilst the experiment was not designed to test an attributional model, it suggests that within experiments of this kind, i.e. tightly defined by the experimenter, there may be no need for attributions to be formed. Attributions may be more important in the clinical interpretation of helplessness where the situation is not defined by the experimenter. There is also the possibility that the attributional check list used in this study was ineffective.

Finally, whilst these concluding remarks have been in support of learned helplessness there is still the fact that most human experiments involve experimenter induced failure. The second experiment defined failure by means of a set of operations and these were not found to be significant determinants of deficits. The points made by Coyne et al. (1980) are still valid and perhaps failure defined operationally in a different way would be effective in the production of learning deficits. This may be a necessary feature of this kind of research as failure is a

subjective experience and may not be defined easily into a set of experimental operations. It is notable that many unsuccessful attempts have been made to demonstrate helplessness using non aversive outcomes. This suggests that whilst negative affective change does not necessarily cause deficits (as shown in study 2) there is something special about a negative outcome. So, even though these results are construed as being supportive to learned helplessness theory, there remain other possibilities that could demonstrate failure to be the causal factor in human helplessness. This seems to be a central problem in attempting to resolve many of the crucial issues in learned helplessness research, namely, to define the cognitive concepts in terms of experimental parameters. For example, to manipulate attributions and to assess experimentally the behavioural consequences of these manipulations. This is a problem as it is difficult to assess attributions as often self reports concerning negative outcomes are distorted for various reasons. These may be, saving face in front of the experimenter, or to preserve self esteem. Whilst it could be argued that these are the important attributional features there is no clear way at present to establish this. It is quite likely that subjects have a realistic attribution concerning the reason for their performance and subsequently deny this for the motives mentioned previously. It is difficult to assess whether the original realistic attribution or the subsequent denial will be reflected in the subjects behaviour.
It is possible that these problems are beyond the capabilities of experimental psychology as we do not have sufficient means to disentangle these effects experimentally.

The second experiment has shown that manipulating instructional set in order to induce the perception of failure causes negative mood changes but does not cause learning deficits. A logical extension of this would be to attempt to discover a manipulation which involved non control but caused a positive mood change or one which involved control but caused a negative mood change. As all combinations of controllability and affective state would be involved, one would have greater confidence that it is the non control that causes the learning deficits and that the negative mood changes were parallel behaviours as opposed to an integral part of a causal pathway resulting in learning deficits. It is through methods like this that other issues may be resolved, such as whether an attribution is an appraisal of a behaviour, a cause of behaviour, a result of behaviour or possibly any combination of the three. Perhaps resolution is too definite as it seems that many issues arising from recent research in learned helplessness may prove to be beyond experimental clarification. This is due to the lack of precision in defining the more cognitive terminology so far. It is not sufficient to designate concepts as hypothetical constructs without precise definition and rigorous attempts at validation. Issues such as those raised by Coyne et al. (1980) regarding failure as opposed to uncontrollability are relevant here as it is not sufficient to subsume failure under helplessness but it must be evaluated critically as an alternative.

In conclusion, the studies provide support for a helplessness model which proposes a direct pathway between uncontrollability and learning deficits. Negative mood change seems to be a parallel behaviour in this case and not a cause of learning deficits, but the role of failure in learned helplessness needs further evaluation.

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APPENDIX

INSTRUCTIONS TO SUBJECTS IN THE PASSIVE CONTROL CONDITION OF THE FIRST STUDY

From time to time a loud tone will come on for a while. Please sit and listen to it, and press the button 4 times when the tone comes on. The red light, indicating the end of the trial will momentarily flash on when the tone stops.

During the course of the experiment, please move as little as possible. The tones will start in a couple of minutes.

Have you any questions ?

INSTRUCTIONS TO SUBJECTS IN THE CONTROL AND NON-CONTROL CONDITIONS OF THE FIRST STUDY

This is an experiment which has been designed to investigate reactions to a slightly unpleasant noise. The majority of experiments which have been carried out usually involve presenting a series of tones over headphones and when a particular response is made on a control box the noise stops immediately. In real life our control over unpleasant events is seldom this immediate, and this experiment will help to show us how people react to less direct control over events in the laboratory.

You will be presented with a series of tones over the headphones. The length of the tones can be controlled by making a particular response on the control box whilst the noise is on. Responses made during the intervals will have no effect. You can make as many responses as you like whilst the noise is on and if you fail to discover the correct response a red light, indicating failure will light up on the display panel at the end of the trial. When you have discovered the correct response, for example, flipping the switch over once and pressing the button twice a green light will light up on the display panel immediately so that you will know what is the correct response.

The experiment is under computer control and when you have made the correct response the computer programme is interrupted and the duration of the noise is reduced. So, once you have made the correct response the Green light will come on immediately but the noise will continue for a while. So you will know that you have done the correct thing but the effect of your action will be delayed.

You are potentially in control of how much noise you will receive. The tones will begin in a couple of minutes and I would like you to discover what the correct response is and also to respond as quickly as possible so that you minimise the amount of noise you receive.

Please use your right hand only and try to move as little as possible.

Have you any questions ?

POST EXPERIMENTAL INTERVIEW FOR SUBJECTS IN THE FIRST STUDY

Name _____ Subject No. _____

1. What was the purpose of this experiment and what were you supposed to do ?

2. Rate how you perceived the noise:-

   a) during the first few trials of the experiment

   pleasant 1 2 3 4 5 6 7 unpleasant

   soft 1 2 3 4 5 6 7 loud

   b) during the last few trials of the experiment

   pleasant 1 2 3 4 5 6 7 unpleasant

   soft 1 2 3 4 5 6 7 loud

3. Rate how you perceived the red light

   a) the first few times it occurred

   pleasant 1 2 3 4 5 6 7 unpleasant

   encouraging 1 2 3 4 5 6 7 discouraging

   b) the final few times it occurred

   pleasant 1 2 3 4 5 6 7 unpleasant

   encouraging 1 2 3 4 5 6 7 discouraging

4. Rate how you perceived the green light

   a) the first few times it occurred

   pleasant 1 2 3 4 5 6 7 unpleasamt

   encouraging 1 2 3 4 5 6 7 discouraging

   b) the final few times it occurred

   pleasant 1 2 3 4 5 6 7 unpleasant

   encouraging 1 2 3 4 5 6 7 discouraging

5. How determined were you to acquire the correct response on the noise task ?

   a) at the beginning of the experiment

   very determined 1 2 3 4 5 6 7 not at all determined

   b) at the end of the experiment

   very determined 1 2 3 4 5 6 7 not al all determined

6. How determined were you to solve the anagrams ?

very determined 1 2 3 4 5 6 7 not at all determined

7. Thinking back to the experiment, did you believe that your responses were effective (or could have been effective) in reducing the duration of the noise ?

YES / NO

How certain are you of this ?

completely certain 1 2 3 4 5 6 7 just guessing

8. At what time during the experiment did you form an opinion about the effectiveness of your responses on the duration of the tone ?

a) at the beginning
b) half way through
c) at the end
d) during this interview
e) not at all

9. During the experiment did you ever have the idea that its purpose might be something other than I was telling you ?

YES / NO

If so, what ?

When did you have this idea ?

a) at the beginning
b) halfway through
c) at the end

10. Have you ever taken any courses in psychology ?

11. Do you read psychology books or journals ?

12. Please make any other comments that you feel might help us understand your reaction to this experiment.

INSTRUCTIONS TO SUBJECTS IN THE NO SET CONDITION OF THE SECOND STUDY

This is an experiment in which we hope to get some information about the relationship between speed of responding and physiological activity under a noise stress, to intelligence as measured by a simple pencil and paper test. We think that the speed which people can do a simple button pushing task may reflect a general pattern of activity in the nervous system, and that a person's performance on an IQ test is similarly dependent on a general activation of their nervous system.

You have already completed a verbal IQ test and now I would like you to do the button pushing experiment so that we can measure your nervous system activity by means of the electrodes I have attached.

You will be presented with ten bursts of unpleasant noise over the headphones and you will be able to turn the noise off if you manage to press the button on the control box enough times within a preset time period. First of all I shall give you a sample of the noise so that you will know what to expect.

In this experiment we have preset a criterion so that you will need to push the button a certain number of times within a specified period before the noise stops. As soon as you have pressed the button the required number of times the noise will stop and a green slide will be shown on the screen to let you know that you have succeeded. If you fail to press the button enough times, a red slide indicating failure will be shown on the screen and the noise will be stopped automatically. It is important for you to press the button as quickly as possible.

I will now show you the slides so that you know what to expect.

INSTRUCTIONS TO SUBJECTS IN THE POSITIVE SET CONDITION OF THE SECOND STUDY

This is an experiment in which we hope to get some information about the relationship between speed of responding and physiological activity under a noise stress, to intelligence as measured by a simple pencil and paper test. We have gathered a lot of evidence from previous research which suggests that a person's reaction time is a very good index of how intelligent they are. We think that the speed which people can do a simple button pushing task may reflect a general pattern of activity in the nervous system, and that a person's performance on an IQ test is similarly dependent on a general activation of their nervous system. You have already completed a verbal IQ test and now I would like you to do the button pushing experiment so that we can measure your nervous system activity by means of the electrodes we have attached.

You will be presented with ten bursts of unpleasant noise over the headphones and you will be able to turn the noise off if you manage to press the button on the control box enough times within a pre-set time period. First of all I shall give you a sample of the noise so that you will know what to expect.

We are nearly ready to start, but first of all I would like to give you some information about the research. We have collected a lot of data on this relationship between reaction time and intelligence and we have found that it is very reliable. In this experiment we have preset a criterion so that you will need to push the button a certain number of times within a specified period before the noise stops. As soon as you have pressed the

button the required number of times the noise will stop and a green slide will be shown on the screen to let you know that you have succeeded. If you fail to press the button enough times, a red slide indicating failure will be shown on the screen and the noise will be stopped automatically.

On the basis of our research, we can predict fairly precisely what you are capable of doing on the noise task and we have set the criterion above the level that most people can possible do. So, it is very important for you to press the button as quickly as possible. If you succeed and manage to get the green slide on, we will increase the number of button pushes on the next trial to make it more difficult. I would like to emphasize that if you succeed you are doing extremely well, much better than the majority of subjects. If you fail and get the red light indicating failure - do not worry as most people are unable to do the task and it is not a direct reflection of your ability but confirms that we have set the criterion too high.

I will now show you the slides so that you know what to expect.

INSTRUCTIONS TO SUBJECTS IN THE NEGATIVE SET CONDITION OF THE SECOND STUDY

This is an experiment in which we hope to get some information about the relationship between speed of responding and physiological activity under a noise stress, to intelligence as measured by a simple pencil and paper test. We have gathered a lot of evidence from previous research which suggests that a person's reaction time is a very good index of how intelligent they are. We think that the speed which people can do a simple button pushing task may reflect a general pattern of activity in the nervous system, and that a person's performance on an IQ test is similarly dependent on a general activation of their nervous system. You have already completed a verbal IQ test and now I would like you to do the button pushing experiment so that we can measure your nervous system activity by means of the electrodes we have attached.

You will be presented with ten bursts of unpleasant noise over the headphones and you will be able to turn the noise off if you manage to press the button on the control box enough times within a pre-set time period. First of all I shall give you a sample of the noise so that you will know what to expect.

We are nearly ready to start, but first of all I would like to give you some information about the research. We have collected a lot of data on this relationship between reaction time and intelligence and we have found that it is very reliable. In this experiment we have preset a criterion so that you will need to push the button a certain number of times within a specified period

before the noise stops. As soon as you have pressed the button the required number of times the noise will stop and a green slide will be shown on the screen to let you know that you have succeeded. If you fail to press the button enough times, a red slide indicating failure will be shown on the screen, and the noise will be stopped automatically.

On the basis of our research, we can predict fairly precisely what you are capable of doing on the noise task and we have set the criterion below the level that most people can easily do. But, it is still important for you to press the button as quickly as possible. If you succeed and manage to get the green slide on, we will increase the number of button pushes on the next trial to make it more difficult. I would like to emphasize that if you succeed you are only doing moderately well - as well as most people can do. If you fail and get the red light indicating failure - then it is a direct reflection of your ability and you should be able to do better.
I will now show you the slides so that you know what to expect.

INSTRUCTIONS FOR THE ANAGRAM TEST TASK

In this experiment I would like you to solve some anagrams. As you know, anagrams are words with the letters scrambled. The problem for you is to unscramble the letters so they form a word. The letters will be projected on the screen in front of you. When you have found the word, please say out loud what it is. If you have not discovered the word within 100 seconds we will move onto the next word.

There could be a pattern or principle by which you can solve all the anagrams, but that's up to you to figure out. Again, during this part of the experiment please move as little as possible.

POST EXPERIMENTAL QUESTIONNAIRE FOR SUBJECTS IN THE CONTROL CONDITION OF THE SECOND STUDY

NAME: _____ NUMBER: _____

DATE: _____

Please answer the following questions which relate back to the experiments you have completed. Please think carefully about your answers so that they are an accurate reflection of the way you really feel.

1) Place a tick beside ONE of the statements below which you think best describes the reason why you did well on the noise task.

   a) you have the ability to do this particular test well.

   b) you have the ability to do psychology tests in general well.

   c) you were in a good mood when you did the test.

   d) you like doing psychology tests.

   e) psychology tests in general are informative so you tried hard to do well.

   f) this particular test may be informative so you tried hard to do well.

   g) today was a lucky day for you.

   h) it was good luck that you were given a test that you could do.

2) Please indicate below how important this experimental situation was to you.

   a) Would it have bothered you if you had failed on the noise task ? YES/NO

   b) Did you feel that it was important for you to do your best

      i) on the noise task ? YES / NO
      ii) on the anagrams ? YES / NO

3) Did you believe what you were told about the experiments at the beginning ? YES/NO

   If not, what did you think the experiments were all about:

4) Which aspect of the noise task was more important to you :

   a) that you were able to control the noise

   or b) that you had done well on a psychology test.

5) Did you expect to do well on the anagram task because you had done well on the noise task ?

YES / NO

6) What was the pattern needed for rearranging the anagram letters to get the correct solution ?

7) Rate the noise on the scales below:

Extremely pleasant ——————————— Extremely unpleasant

Extremely soft ——————————— Extremely loud

8) How hard did you try to succeed on the noise task ?

Very hard ——————————— Not at all

9) How hard did you try to solve the anagrams ?

Very hard ——————————— Not at all

10) Rate your experience with anagram solving, bearing in mind whether you do crossword puzzles etc.

Very experienced ——————————— No experience at all

11) Have you ever taken any psychology courses ?

YES / NO

12) Do you read psychology books or journals ?

YES / NO

13) Please make any other comments that you feel might help us to understand your reaction to these experiments ?

POST EXPERIMENTAL QUESTIONNAIRE FOR SUBJECTS IN THE NON CONTROL CONDITION OF THE SECOND STUDY

NAME: _____ NUMBER: _____

DATE: _____

Please answer the following questions which relate back to the experiments you have completed. Please think carefully about your answers so that they are an accurate reflection of the way you really feel.

1) Place a tick beside ONE of the statements below which you think best describes the reason why you did not do well on the noise task.

   a) you lack the ability to do this particular test.

   b) you lack the ability to do psychology tests in general.

   c) you were in a bad mood when you did the test.

   d) you dislike doing psychology tests.

   e) psychology tests in general are uninformative so you didn't try hard.

   f) this particular psychology test is uninformative so you didn't try hard.

   g) today was an unlucky day for you.

   h) it was bad luck that you were given a test that you could not do.

2) Please indicate below how important this experimental situation was to you.

   a) did it bother you that you had failed on the noise task ? YES / NO

   b) did you feel that it was important for you to do your best

      i) on the noise task ? YES / NO

      ii) on the anagrams ? YES / NO

3) Did you believe what you were told about the experiments at the beginning ? YES / NO

   If not, what did you think the experiments were all about :

4) Which aspect of the noise task was more important to you:

   a) that you were unable to control the noise.

   or b) that you had failed on a psychology test.

5) Did you expect to do badly on the anagram task because you had done badly on the noise task ?

YES / NO

6) What was the pattern needed for rearranging the anagram letters to get the correct solution ?

7) Rate the noise on the scales below:

Extremely pleasant ——————————— Extremely unpleasant

Extremely soft ——————————— Extremely loud

8) How hard did you try to succeed on the noise task ?

Very Hard ——————————— Not at all

9) How hard did you try to solve the anagrams ?

Very hard ——————————— Not at all

10) Rate your experience with anagram solving, bearing in mind whether you do crossword puzzles etc.

Very experienced ——————————— No experience at all

11) Have you ever taken any psychology courses ?

YES / NO

12) Do you read psychology books or journals ?

YES / NO

13) Please make any other comments that you feel might help us understand your reaction to these experiments ?

FEED BACK SLIDES USED IN SECOND STUDY

FAILURE

a YOU HAVE DONE VERY POORLY

PLEASE TRY HARDER

FAILURE

b YOU HAVE ONLY JUST FAILED

DON'T WORRY - TRY AGAIN

SUCCESS

c YOU HAVE ONLY JUST SUCCEEDED

PLEASE TRY HARDER

SUCCESS

d YOU HAVE DONE VERY WELL

PLEASE KEEP IT UP

KEY TO FEED BACK SLIDES USED IN SECOND STUDY

a)	NON CONTROL	NEGATIVE SET	WHITE LETTERING ON RED SLIDE
b)	NON CONTROL	POSITIVE SET	
c)	CONTROL	NEGATIVE SET	WHITE LETTERING ON GREEN SLIDE
d)	CONTROL	POSITIVE SET	